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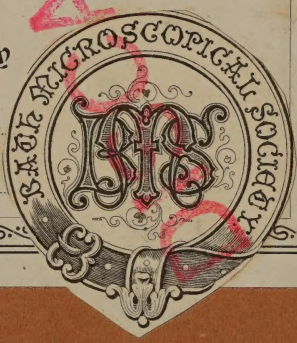

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ARCHIVES

OF

MEDICINE:

A RECORD OF PRACTICAL OBSERVATIONS AND ANATOMICAL AND CHEMICAL RESEARCHES CONNECTED WITH THE INVESTIGATION AND TREATMENT OF DISEASE.

EDITED BY

LIONEL S. BEALE, M.B., F.R.S.

VOL. IV.

With Original Papers and Memoirs.

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NOTE TO PAR. ON PP. 276-7, VOL. IV.

"Subsequent observation has confirmed me in the belief that the structure described in this paragraph consisted of blood-vessels, surrounded by cancer-cells, together with layers of blood-globules."

ARCHIVES

OF

MEDICINE.

VOL. IV.

FATAL CASE OF INHALATION OF CHLOROFORM.

By George Robert Cubitt, of Stroud.

The following will be found to differ but little from numerous cases already placed on record; still it seems to me to present certain points of interest which may tend to a more precise appreciation of the mode, or at least of one of the modes, in which chloroform produces death; and were it otherwise, I should not the less feel it my duty in the present state of our information on the subject, to offer for publication the details of any case in which the inhalation of that agent had led to a fatal issue.

T. W., æt. 22, by calling a chimney-sweep, was received from the Union House, into the Stroud Hospital, on October 2, 1862, for active disease of the right knee joint, the result of an injury some three months previously. At the end of three weeks he was seen in consultation by all the members of the medical staff, who unanimously decided that the limb should be submitted to amputation, while the patient was under the influence of chloroform. At his own earnest request, October 23rd was named as the earliest possible date for that purpose. He had a better night than usual on the 22nd; at 7.45 the following morning had a light breakfast, consisting of tea, bread and butter, and one egg; and at 11 a.m., the hour fixed for the operation, was in excellent spirits. He was above the average height, of sallow complexion, with dark, straight, and wiry hair; was naturally of a spare habit, but of late had become thin and emaciated, and looked much more than his age. His thoracic

and abdominal viscera had been carefully examined and pronounced sound; nor did he seem to suffer from any disease save that of the knee joint. This was hot, swollen, and acutely tender, especially upon the slightest movement. There were openings, both upon the inner and outer aspects of the joint, which had been artificially made for the evacuation of matter, which during his residence in the hospital had been very profuse. The pulse had never been under 100, but there had been no diarrhœa or night sweats; the appetite, though uncertain, had on the whole been tolerable; his nights had been bad, and occasionally, he had been troubled with a loud barking cough, attended with slight mucous expectoration. Taking into account the gravity of the local disease, the constitutional symptoms might be considered as below the average; certainly they could not be regarded as extreme.

As it had been arranged to operate in the ward—which was empty—it was resolved to administer the chloroform to the patient while in the recumbent position in bed. The inhalation was commenced, in the presence of all the members of the staff, at about 11.15 a.m., that is, just three and a half hours since the last meal. It was administered by my friend Dr. Paine, who has been in the habit of similarly officiating on such occasions, and has otherwise had large and varied experience in the employment of this agent. The inhaler employed was that of Dr. Snow, and the chloroform was from a sample which had been made use of by the same gentleman in the same institution only 24 hours previously, while I was removing a small tumour from the leg. While Dr. Paine took especial note of the patient's respiration and of his general aspect, and satisfied himself from time to time as to the state of his pulse at the wrist, another of my colleagues,—Mr. Wethered,—never took his finger from the radial artery of the opposite side, while I myself was seated at the foot of the bed, watching the pulsations of the posterior tibial. There is the most entire agreement of opinion between us, that up to the moment of removing the patient from his bed, the pulse never so much as once failed or faltered in the slightest degree. At the commencement of the inhalation the pulse was about 120; it subsequently fell a few beats as the chloroform began to affect the patient, but rather gained than not in power; it again rose for a short period while the patient was exhibiting some violence, and once more subsided without losing power, as the patient became tranquil. It was generally remarked that the patient was very slow to evince the effects of the anæsthetic even in its first and earliest degree; but this was thought to be explained by the mouth-piece not being

quite large enough to embrace the entire mouth, so that the vapour of chloroform was additionally diluted by that amount of atmospheric air, which entered the lungs through the angles of the mouth, without passing through the narcotising agent at all. The first appreciable effects of the chloroform were manifested in the production of a certain amount of hysterical excitement, very much in accordance with the patient's natural temperament, which was of an unusually demonstrative character; this became exaggerated into a state of "crying drunkenness" as the inhalation proceeded, and was shortly followed by a brief period of violence, during which the patient had to be restrained; it was in this stage that the pulse rose a little, and the countenance became somewhat flushed, but there was nothing approaching stertor, or convulsive action; the strugglings were those of a drunken man, directed to a definite end, namely, that of getting rid of the inhaler; and the condition of pulse and countenance were simply the proportionate results of physical exertion; so far was there from being present any excess of narcotism at this stage, that the patient was still able to express himself in articulate language, exclaiming, "Take it away! take it away," &c. This state of matters was shortly succeeded by a tranquil sleep, during which the face was perfectly natural, the respiration entirely regular and unimpeded, and the pulse about 100, and of fully equal force as immediately before the commencement of the inhalation. I now adjusted the tourniquet previous to tightening; the inhaler was removed, and I left my colleagues to lift the patient from the bed to the table. So far was the insensibility even now from being profound, that there was some apparent evidence of pain while the limb was being disturbed for the adjustment of the tourniquet; and thinking more chloroform would be needed before the completion of the operation, Dr. Paine was replenishing the inhaler while the patient was being moved. During the same short interval I had turned round to see that all the necessary instruments were at hand, when both that gentleman and myself were attracted by a noise, as if our patient were about to vomit. We turned round, and saw at a glance that he was either actually dead or dying. The face was pale and tranquil; the eyes up-turned, with pupils dilated; there was no froth about the mouth. The pulse was entirely gone; the breathing completely suspended; not the faintest sound could be heard at the heart. The pillows were immediately pulled from beneath the patient's head; the strongest liquor ammoniæ applied to the nostrils; and the tongue seized with forceps, and forcibly pulled forward. This all being without result, I immediately thrust a scalpel through the crico-

thyroid membrane, making a free opening; not a drop of blood followed the knife; but almost immediately afterwards the patient fetched a deep sighing inspiration, but I could hear no air enter the trachea. I at once applied my mouth to the opening, and artificially inflated the lungs, or rather I should say forcibly expired into the trachea, for the proceeding was not attended with any visible expansion of the chest. A flexible catheter was next introduced through the wound into the trachea, and inspiration and expiration attempted alternately. In the meanwhile brandy had been poured into the patient's mouth. There were no signs of deglutition, but the brandy neither escaped through the mouth nor produced choking. Presently there was another ineffectual attempt at inspiration, that is to say, the patient's mouth opened and closed, and there was a certain amount of movement about the neck and throat, but the walls of the chest remained motionless, and no sound could be heard as of air passing into the chest. The ammonia and brandy had been continued, and the surface had been dashed with cold water, but without satisfactory results; no true inspiration had been accomplished, nor had the heart responded in the faintest degree to our efforts. A wheel galvanic battery, after the American pattern, was quickly got to work, one pole being applied to the cervical spine, and the other alternately to the region of the heart and to the epigastrium, while my colleagues assiduously persevered in postural artificial respiration, after the manner of Marshall Hall. Two more ineffectual attempts at inspiration, like those already described, followed at lengthening intervals, and of successively diminishing power, when even these ceased, and we were at last obliged to acknowledge to ourselves that our patient was actually dead. Not a pulse at the wrist had been felt, nor a sound at the heart heard since the first moment of our taking the alarm.

There is some obscurity as to the exact amount of chloroform employed; it was poured into the inhaler from a graduated bottle, by reference to which, of course, the gross quantity absorbed would be ascertained; but it has been seen that the inhaler, after it was replenished, was not employed; and as the amount by which it was replenished was not measured separately, while to the quantity left in the inhaler must be added that lost by evaporation, and what was absorbed by the bibulous paper, an approximation only to the actual amount employed can be arrived at. Dr. Paine estimates it as certainly under three drachms; of course a very much smaller quantity than this entered the lungs, while the time employed in the inhalation before the production of any anæsthetic effects was unusually long.

Post Mortem.—Three hours and three quarters after death the body was examined in the presence of the medical staff, and of a gentleman—Mr. Blagden—unconnected with the hospital.

The left pupil was found widely dilated; the right contracted. In each eye there was some vascularity—scarcely amounting to extravasation—around the lower circumference of the cornea, and a little overlapping its margin.

Upon opening the thorax, the heart and lungs as seen in situ, appeared normal in their relations to each other, and to the walls of the chest. The base of the right lung was attached to the diaphragm by old adhesions, and a few fibrous bands of similar character and date, passed, here and there, from the posterior border of the same lung to the parietal pleura. There was no fluid in either pleural cavity. A ligature having been put round the large vessels, the heart and lungs were removed from the chest. Anteriorly and laterally, the pulmonary surface was much paler than ordinary, and behind presented less than the usual amount of post-mortem congestion. The pulmonary tissue was universally crepitant, and without a trace of emphysema, or of any abnormal deposit; there was no approach to congestion, and the cells and tubes were unobstructed by mucus. There was no fluid in the pericardium; no adhesions; no fibrous patch upon the heart; no deposit of fat. The heart was of natural size; the left auricle seemed empty; the left ventricle, firmly, and even *vehemently* contracted; the right auricle was full; the right ventricle distended. Upon opening the cavities of the organ, the blood in the right auricle was found partly coagulated, and of the consistence of warmed currant jelly. The amount of blood in the right ventricle was very considerable; it was partly fluid and partly coagulated, and presented large coagula of decolorized fibrine. The pulmonary artery was empty; the pulmonary veins and left auricle were perfectly empty of blood; the left ventricle not only empty, but so firmly contracted, as scarcely to leave the trace of a cavity. The degree of contraction was quite remarkable. On both sides, the great vessels, the auriculo-ventricular orifices, and the valvular apparatus were entirely healthy. The tissue of the heart throughout was well nourished, and beyond all suspicion of fatty or other deposit. The liver was gorged with blood, and occupied more than its normal space in the abdomen, but its tissue was entirely healthy. The spleen was congested, but in a minor degree. The stomach contained a considerable quantity of fluid, the patient's last meal not having escaped from its cavity. The intestines, a little distended with flatus, were otherwise empty. Both the kidneys were gorged with blood, large, heavy,

and a little mottled. The left certainly suggested to me the notion of its being in the very earliest stage of Bright's disease, but this opinion was not shared by any of my colleagues, who regarded the appearances as those of simple congestion. I mention the discrepancy as a matter of fact; but the discrepancy itself will show that supposing the existence of disease, it was not in a degree to have any practical bearing on the cause of death. The brain and its investments were most carefully examined, but without discovering a vestige of disease, whether as regards texture or vascular supply. The spinal cord was not examined.

Now, this case, as it is certainly one of painful interest, so from its very simplicity, from the absence, I mean, of all complications, does it appear calculated to render somewhat more precise our notions of the manner in which chloroform may produce death. Here is a patient in the best and most vigorous years of life, entirely free from any disease but the local affection for which it had been proposed to operate, undeniably killed by the action of chloroform, administered after an orthodox fashion, within the generally allowed limits, and without the neglect of any ordinarily prescribed precaution; nor was the fatal issue, so far as I could see, in any degree retarded by the employment, with promptitude and energy, of every means suggested as likely to be of service in such cases. A more than ordinary amount of time, owing, as I believe, to an unusual degree of dilution of the vapour by atmospheric air, had been occupied in developing the anæsthetic effects of chloroform; up to a certain moment everything had been satisfactory as regards the effects of the agent: there had been no deep oppression of the nervous centres, as evidenced by stertor and convulsion; the circulation was well maintained; the respiration regular, tranquil, and altogether unembarrassed; another moment, and the heart's action is suspended, never to be resumed, while three or four attempts at inspiration at lengthening intervals, alone prevent us from describing death as instantaneous. These attempts at inspiration, however, after the heart had finally ceased to beat, sufficiently indicated the efficient cause of death to have been located in the heart rather than in the lungs; and accordingly, upon a post-mortem examination, the lungs are found to be perfectly healthy, free from congestion, and well supplied with air. Most assuredly the death here is not death from asphyxia. Whenever the right ventricle may last have delivered its contained blood to the lungs, these organs have duly passed on such blood to the left side of the heart, and have waited in readiness to aerate a further supply. That after death they should have been

found bloodless and air-*full* demonstrates that such further supply of blood never reached them. Wherefore? When having examined the lungs we pass on in the order of circulation, to the left side of the heart, we find the pulmonary veins empty, the left auricle empty, the left ventricle empty; whenever, therefore, the right ventricle may have delivered its blood to the lungs, that blood has passed on in due succession to the left side of the heart, and from thence through the aorta into the systemic circulation. We must therefore look further for the arrest of the circulation. We accordingly arrive at the right side of the heart, and find its auricle moderately full. So far, therefore, the blood has travelled; but beyond this, we find the *ventricle* not only full but distended; it has at any rate received its full supply through the auricle of its own side; but beyond this ventricle we find no more blood; the arrest of the circulation, therefore, whatever its proximate cause, has for its seat the right ventricle of the heart. And this, I believe, is exactly the state of matters wherever death has resulted from the inhalation of chloroform; the left cavities of the heart have uniformly been in a condition of comparative or absolute emptiness; the right cavities more or less full and distended. The condition of the lungs as regards the supply of blood has varied, however; where the circulation has continued after the respiration has ceased, that is, where the death has been by asphyxia, the lungs have been found congested, the remora in the circulation having the pulmonary capillaries for its seat; but where, as in the present case, the respiration has continued after the circulation has ceased, the lungs have been found bloodless, the remora in the circulation having been localised in the right ventricle, that is, where the death has been by cardiac syncope. Now where, as in cardiac syncope, the lungs have not had their supply of blood renewed from the right ventricle, we can only suppose it due to one of two causes,—there has either been a spasm of the pulmonary artery, which the contractions of the right ventricle have not been able to overcome; or, secondly, the ventricle has not been in a condition to contract upon its contents at all; the one would be death by spasm, the other would be death by paralysis of the heart. The first hypothesis, I think, we may at once dismiss from our consideration as entirely gratuitous, and altogether unsupported by analogy. We are therefore left to deal only with the latter assumption, that death was due to paralysis of the right ventricle. We may remark, *en passant*, that the condition of the vascular system behind the heart as contrasted with that before it, confirms the notion of the obstruction to the circulation being seated in its right

cavities. In front of these cavities, that is to say, in the lungs, left heart, and systemic capillaries, we find an absence of blood; while behind the right heart and in proportion to its neighbourhood to it, that is to say, in the liver, in the spleen, in the kidneys, we find a superabundance of blood. There seems one exception to this statement, there was no cerebral congestion; but perhaps it is not too much to suppose that the blood in the right auricle was mainly due to post-mortem gravitation from the parts within the cranium. But admitting death to have been the result of paralysis of the heart, it remains to inquire into the proximate cause of such paralysis, the *rationale* of its production. Now, when we venture to speak of paralysis in this case, we seem at once to be admonished by the contrasted condition of the two sides of the heart. Nothing on the left side betokens any loss of power; here the auricle has performed its duty, while the ventricle by its very condition—empty, hard, and rigid—demonstrates that its very last action was one of intense energy. The idea of paralysis on one side seems contradicted by the very spectacle of power on the other. The intervening moment between life and death—if we may be allowed for the purpose of illustration to use such a phrase—corresponded with the heart's last systole; on the left side this was accomplished with the greatest energy, and the contents of the ventricle were successfully delivered to the systemic capillaries; on the right side, the ventricular contraction failed to follow the preceding diastole, and the cavity remained full at the moment of death, the pulmonary capillaries being empty. Now this contrasted condition of the two sides of the heart, at once relieves the nervous centres of any suspicion of complicity in the effect produced. Any agent exercising a central operation, could not possibly have a one-sided effect for its consequence; both sides of the heart deriving their nervous supply from the same source, an agent operating upon this source could not produce excess of power upon the left, and deficiency of power upon the right side; the paralysing agent, therefore, must be sought for in the paralysed part itself, within the sphere of which its influence must be confined. Now, that the vapour of chloroform is a direct sedative to the heart when brought into contact with its muscular tissue, is proved by the experiments of Dr. Snow, who states that he has frequently arrested the action of the heart in animals, opened immediately after apparent death, by blowing the vapour of chloroform upon it. It has also been again and again ascertained that when chloroform has been inhaled, its vapour is absorbed into the blood, and can be detected at any point of the circulation.

It is clear, therefore, that chloroform—as chloroform—arrives at the right side of the heart with the column of returning venous blood, after its absorption into the circulation during inhalation; and there is as little reason to doubt, that under favouring circumstances, whether these consist in undue concentration of the agent, or in unusual susceptibility of the subject, that the presence of chloroform in the right side of the heart, may arrest its contractions, as in the experiments of Dr. Snow. That it may be present in the left cavities of the heart without producing the same result, does not invalidate this view, but only goes to prove that a smaller quantity of the agent, admixed with venous blood, as on the right side, suffices to destroy its power of stimulating the heart to contract, than is required when mingled with pure arterial blood as on the left side, a view entirely supported by the experiments of Dr. Glover, in 1842, (as quoted by Dr. Snow,) who found that: "in animals killed by injecting chloroform into the jugular vein, the irritability of the heart was destroyed whilst this organ retained its irritability, in those which were killed by injecting it into the arteries, stomach, or peritoneum." Whether the chloroform fails to paralyse the left side of the heart, because its effects are suspended by the higher oxygenation of arterial blood, and succeeds in paralysing the right side by reason of the carbonization of venous blood; or whether the presence of chloroformic vapour, beyond a certain proportion in the circulation, so far suspends the action of the various depurating organs, that the blood, upon its return to the right ventricle, ceases to be a sufficient stimulus, on this account, to its contraction, must be left for further inquiry and experiment to determine. If the simple presence of oxygen in arterial blood be the efficient cause of the retained irritability of the left side of the heart, and it should be subsequently proved that this is not incompatible with the production of the true anæsthesia required, it suggests the practicability of diminishing the danger of chloroform by diluting its vapour with some aeriform fluid, more highly oxygenated than common atmospheric air.

I think it is sufficiently evident that the state of matters we have been considering, that of paralysis of the heart, the consequence of a poisonous proportion of chloroform in the right ventricle, is very little remediable by treatment; indeed, when well established, it is simply an amplification of the word "death." It is little remediable, because unless successfully treated almost at the very moment of development, the time for all treatment has passed away. We must remember too, that the immediate effect of the presence of the poison in

sufficient quantity, is to arrest the very action by which alone that poison can be removed; while, supposing our treatment to be so far successful as to produce a ventricular contraction which shall empty its cavity of the modicum of the poisoned fluid, it is only to have it replaced during the diastole, which succeeds, by another not necessarily much more pure than the last. It is in this respect that such a case differs so emphatically from simple syncope; here, if we can only excite so much as one more effort of the circulating forces, this first success becomes the parent of a second, inasmuch as it contributes in some proportionate degree that healthy stimulus which the heart is needing; but in the case we are considering, the natural stimulus itself is poisoned, and life must almost necessarily become extinct before our temporary success can effect its purification. We must rather look, therefore, to preventive treatment, endeavouring by a more precise appreciation of the effects of chloroform during the earlier stages of inhalation, to anticipate the moment of danger, and so to come to the assistance of the heart before it actually fails, and while it only falters. The means of arriving at this knowledge with precision and certainty have still, I think, to be devised. The mere computation of the beats of any particular artery, or even a moderately careful attempt to estimate the volume of such pulsation, would not seem to be sufficient; at least, in the present case, both together signally failed to yield any intimation of approaching danger. It may be urged that this and similar cases are instances of s u d d e n death, and that in such cases there is a necessary absence of all manifestations at which precaution can take alarm. But, if it be correct to attribute death in this instance to the poisonous accumulation of chloroform in the blood, death cannot be said, in a physiological sense, to have been sudden; time, in some proportion, is necessary to accumulation, and death under such circumstances can only be called sudden, because in the present state of our information, we are without the means of measuring the progress of such accumulation. Now, the pulsations of any given artery, as the radial or the tibial, whether we have regard to their number or their volume, only inform us d i r e c t l y of the condition of the left ventricle and the systemic circulation; of course, i n d i r e c t l y, they inform us also of the condition of the right ventricle and the pulmonary circulation, but certainly with less precision. But we have seen that in this case, at the moment of death, and we know not for how long previously, the state of the right ventricle so far from being the same as that of the left, was directly in contrast with it.

If at the moment of death the left ventricle were acting with intense energy, and the right not acting at all, it is allowable to assume, that at some moment more or less remote from that of death, while the left ventricle was still acting vigorously, the right was already becoming embarrassed, and of this embarrassment, according to the report of the present case, the condition of the pulse at the wrist was not a sufficiently palpable and practical sign. I say a "sufficiently palpable and practical sign," because I do not mean to imply that one-half of the circulation could be affected without having an influence upon the other; of course, the volume of the arterial pulse must be in a ratio with the column of blood reaching the left from the right side of the heart; but it is equally clear that the ordinarily educated finger may fail to detect a diminution of the column in time to avert danger. I am not pretending to point out the necessary means, but simply endeavour to impel the inquiries of scientific observers along this channel. To say so much, may, I am aware, be an unfair exaggeration of the views of both parties, but practically speaking, the administrators of chloroform may be divided into two classes; the one seeks for the intimation of danger in the condition of the respiratory function; and the other concentrates its attention upon the state of the circulation. Both are so far in the right, that death may equally begin at the heart or in the lungs; and I would venture to suggest, that whether the danger lie in the one direction, or in the other, in either case it is possible that its earliest intimation may be found, not in the respiratory or in the circulating functions absolutely and separately, but in the condition of either in its relation to the other. In a condition of health, a certain balance is maintained between the circulation and the respiration, which balance is disturbed by various states of disease; the connection between the pulmonic capillaries and the right ventricle is as direct and intimate as that between the left ventricle and the systemic capillaries; and taking into account the relation between respiration and circulation, it is not unreasonable to conjecture, that the volume, rhythm, and rate of the respiratory action, may be found as trustworthy an index of the condition of the right, as the arterial pulse is accepted to be of the left ventricle; and therefore is it that I humbly venture to discern the avenue to a more precise estimate of the powers of the heart generally during the inhalation of anæsthetics, by a careful application of a previously well-determined system of comparison between the respiratory and circulating acts, rather than by the examination of one separately from the other.

It is clear, that in a case of this kind the prime object in treatment must be the stimulation of the heart; stimuli addressed to the respiratory functions, however essential to the continuance of the heart's action, when once renewed, must not be allowed to take the place of measures directed to the artificial stimulation of the heart itself. In the present case, the lungs were awaiting, not a supply of air, but of blood, which nothing but a contraction of the paralysed ventricle could furnish. The remedy needed, therefore, was that by which such contraction could be excited. It must clearly be one which may act with the greatest amount of energy in the shortest possible time. In such a juncture it does not seem to me that the measures ordinarily in use, or at any rate that those as usually employed, would be at all likely to succeed. These are the indirect stimulants, such as alcoholic and ammoniacal solutions; and what I suppose we may call the more direct stimulation of a galvanic current. However valuable the former may be in keeping up an enfeebled circulation, few I think would rely with great confidence upon their powers of renewing a suspended circulation. Their consideration, however, suggests one point of practical importance,—the extreme necessity of insuring the absolute emptiness of the stomach during the inhalation of chloroform. In this case between three and four hours had elapsed since the last meal, and yet that meal had never passed from the stomach. What effect for good was brandy or wine likely to have in such an emergency, when diluted with the fluid contents of a half filled stomach? To be of any service such remedies must be received into an empty organ, and not only so, but they must be administered in as concentrated a form as is compatible with the ultimate integrity of the mucous membranes with which they come into contact. Where there is no circulation, nothing can be hoped for from absorption into the vessels of a viscus at a distance from the organ to be stimulated; a secondary effect from the immediate impression upon the nerves of the part is all that can be anticipated, and this impression will be in proportion to the concentration, rather than to the quantity of the agent employed. There is something more specious in the employment of the electro-galvanic current; but as generally employed, I believe it will be found even less satisfactory. In the present instance it was found to act with remarkable energy upon the voluntary muscles in the line of circuit between the two poles. Accordingly, as one pole was placed at the epigastrium, or the left mammary region, and the other in the spine, the bellies of the corresponding muscles swelled up in vehement contraction, and it

was hard to believe that the arm which was powerfully drawn across the chest, was only the arm of a dead man. According to the point of contact with one pole of the battery, the other being placed upon the cervical or upper dorsal spine, the various muscles engaged in respiration were thrown into vehement contraction; but all along there was as entire an absence of that co-ordinate muscular action necessary to the expansion of the chest, as of that cardiac contraction which was t h e one thing needful. All the muscular action excited was without, not within, the chest, the necessary current being completed by the superficial, without the intervention of the deep-seated parts, and neither the heart nor the diaphragm was one atom the more directly stimulated into action because of the imposition of a pole of the battery over the seat of the one or the other, than if it had been directed to the extremities of the body. Knowing the intimate connection between the heart and the stomach, it may be fairly questioned whether the reception of a glass of pure brandy into the cavity of the latter would not be far more calculated to rouse the former into activity than the application in the manner described of a stimulus which is quite as effective in exhausting as in resuscitating nervous irritability. Still the effects of an electro-galvanic current in exciting the contractility of the voluntary muscles, naturally suggests the possibility of all striped muscular fibre being within its influence, and dictates the propriety of making an attempt to bring the tissues of the heart as directly within the circuit of the current as that of the superficial muscles. Acupuncture of the heart has before now been practised with impunity; and I do not see why, in such a desperate juncture as that we are now considering, we may not be allowed to penetrate the heart itself with needles especially prepared for that purpose, and connect them with a pole of the battery; still less do I see why, after introducing an æsophageal tube into the stomach, which may equally serve as a means of emptying the organs of its contents and of introducing powerful local stimuli, we may not take advantage of the intimate nervous relations between the heart and stomach, and by introducing the extended pole of a battery through such tube make the most powerful of all indirect appeals to cardiac contractility. I am not wishing to depreciate the value of artificial respiration; in such cases we only i n f e r the condition of the lungs as to supply of blood and air; and even if we are correct in our interpretation of the case, artificial respiration may still be necessary to continue the circulation which by cardiac stimulation we may have been fortunate enough to excite; all I would plead for is, that in every case plainly one of cardiac paralysis, the heart should have the

first thought, the respiration the second; just as where death is imminent from evident asphyxia, air should be looked upon as the prime necessity. The pulling forward of the tongue—this failing, the opening of the larynx—and the method of postural respiration may all become invaluable as the means of perpetuating a first success, or of preventing the occurrence of death after one manner, while we are labouring to prevent its being brought about by another.

I would only add a word touching the appearance of the eyes, as observed after death; I mean the conjunctival vascularity, and the inequality of the pupils. Without attempting to explain the manner in which these phenomena were produced, I can speak to their being post-mortem appearances; the condition of the eyes engaged at the time my peculiar attention, and I can positively testify that when the corpse was removed from the table the pupils were greatly and uniformly dilated, and that there was an entire absence of the vascularity observed after death, unless it were hidden beneath a thin film of secretion which shortly collected over the corneæ.

ABSTRACT OF A PAPER ON ERECTION OF THE PENIS.

By Professor C. Eckhard, of Giessen.

In spite of the many researches made upon this subject since the revival of learning to the present time, its very nature is still unknown. In order to study the phenomena, it seems to me, above all things, necessary to find out in any animal the nerve which is immediately concerned in erection, for by a certain operation the physiologist is able to produce erection in an animal when he chooses. In a paper to be published shortly,* I have described the results of an investigation on this subject but I propose now to give a short abstract of the conclusions. In the first part I have given an historical sketch of all the papers relating to the subject, from de Graaf and Swammerdam, to Kölliker and Kohlrausch. As this part is a mere historical one I shall not refer further to it here. In the second part I acquaint the reader with the most essential facts regarding the structure of the penis of the dog, the animal on which I have exclusively operated. To understand the physiological experiments presently to be mentioned, it may suffice to refer the

* See my contributions to Anatomy and Physiology, vol. iii.

reader to the following data. The corpus cavernosum urethræ is especially developed, whereas the corpora cavernosa penis are merely rudimentary in this animal. The former consists of the trabecular tissue well known to descriptive anatomists. At the posterior part, the trabeculæ form, as in man, the bulbus urethræ, at the anterior part is a second bulb which projects into the glans. The veterinarians consider this second bulb as a part of the glans, but I think with no sufficient reason. I will not, however, enter now into further details, but will only remark, that the anterior bulb swells up when erection takes place, and that at the time of cohabitation it is introduced into the female parts, and by thus becoming still larger, it partly contributes to effect the long coitus which occurs in the dog. In the posterior bulb the trabeculæ are small and thin, in the anterior, they are broader.

The arteries are arranged as in man. Each corpus cavernosum penis receives its arteria profunda, and into the corpos cavernosum urethræ enter two arteriæ bulbosæ with the following peculiarities:—In the posterior bulb each artery gives origin to a certain number of small serpent-like branches which run through the whole thickness of the bulb from the insertion of the urethra towards the outer surface. The smallest twigs lie in the substance of the trabeculæ themselves. At present, we need not study the more minute structure of all these parts, for our investigation is mainly of an experimental character, and may be understood without entering very minutely into anatomical details. In the middle of the corpus cavernosum, urethræ, and in the anterior bulb, the arterial twigs get much smaller and less abundant. From this we may conclude that most probably the two bulbs perform different functions.

As to the veins, the most important are as follows:—

1. The two dorsal veins. Passing under the symphysis they unite, and afterwards divide again, but into two veins of unequal size. The union of the dorsal veins is imbedded in the tendons of two small muscles described by Houston, and called by him the compressores venæ dorsalis.

2. The venæ profundæ and bulbosæ, which require no detailed description.

3. A vein coming from the glans penis and the preputium, which opens into the vena epigastrica externa.

There are on each side two nerves. The first is sufficiently known as the nervus dorsalis, the second requires a special description. In the dog this nerve comes from the

ischiadic plexus, in which it can be traced back to the two or three first sacral nerves. Sometimes, although very seldom, this nerve is divided into two branches. It runs along the vessels destined for the bladder and prostrate gland, and close to these parts it enters the hypogastric plexus. In my paper above referred to, I have given a sketch of its course. The hypogastric plexus gives off small twigs for the bladder, prostate, and the membranous part of the urethra. Those fibres which accompany the latter contain the fibres taking part in erection. These being the same fibres which coming from the ischiadic plexus entered the hypogastric plexus as proved by physiological experiments. They are extremely fine, so that till recently, I could only trace them to the bulbus urethræ which they penetrate to reach the interior of the corpus cavernosum urethræ. The other constituent branches of the hypogastric plexus have nothing to do with erection.

I next proceed to describe my experiments. First, I have paid attention to the dorsal nerve. In one series of experiments I cut it, or rather I took out a short piece in order to prevent any regeneration. In all cases I observed no constant effect whatever. In a second series of experiments in which artificial irritation of the peripheral part of the divided nerve was carried out, negative conclusions were alone arrived at.

Next, I took into consideration the second nerve before described. The mode of operation to get at the nerve conveniently for irritation I will not describe here, for in my paper I have given a sufficiently detailed account. The first experiment led to the observation, that this nerve presides over erection, for I had hardly commenced to irritate it when the corpus cavernosum urethræ began to fill with blood, and in a few moments the anterior bulb appeared as a large and very hard swelling. Many experiments executed in the same way produced the same effect. I have only to add, that if the animal be a young one (about three-quarters of a year or less), or be subjected to castration, erection presents itself in a less degree. Still it is observable, especially when castration was carried out in later years. I need scarcely say that in man erection takes place in eunuchs, for the 6th of Juvenal's Satires gives us sufficient proof of this fact. It may be interesting to mention the further observation, that when the nerve repeatedly mentioned is irritated, evacuation of the liquor prostaticus also takes place. Hence the fibres for erection and those concerned in the process of squeezing out the prostatic fluid are contained in the same peripheral nerve.

Thus we have succeeded in finding means to produce erec-

tion in an animal whenever we desire; ought we not, therefore, to be able to make out the very nature of the phenomenon in question? I confess there is still a question to solve, but I may describe in what erection mainly consists. The reader knows what different and often contradictory statements are made, by both ancient and modern physiologists upon this point. Some believe that during erection, more blood is introduced into the penis, whereas others are of opinion that the swelling is effected if the veins be partly closed in any way. We may decide between these two hypotheses. I laid bare the pudic vein, where it passes the cavum rectoischiadicum, and cut it at a point just beyond that where all the veins of the penis had joined it. Now, I measured the quantity of blood previous to erection and also during this state. In all cases I got a much larger quantity of blood at the time of irritation than in the common state of flaccidity of the penis.* In spite of this all parts of the penis were hard and large. From this observation it follows, that during erection a larger quantity of blood is effused into the trabecular tissue, and that the common reflux of the blood through the veins is by no means interfered with. On the other hand it is accelerated. The experiments are somewhat difficult. They require much time, and, most of all, patience. Happily we may substitute for them the following experiment, which, although not so conclusive, still shows the increased flow of blood into the veins. After having prepared the nerve for a successful irritation we pick out one of the dorsal veins and observe the jet of blood which issues from it; first, when no irritation is produced, and, secondly, when the nerve is irritated. Under these circumstances we observe, that in the former case only a slow flowing of blood is to be seen, whereas in the latter the vein discharges a considerable quantity of blood, quite like an artery.

I believe that the apparatus by which the great quantity of blood is poured into the penis is solely lodged in the posterior bulb of the corpus cavernosum urethræ, for when making openings in it in such a way that they lie in a straight line from behind forwards, during irritation we observe, first, a jet of blood from the opening most posterior; after a little while, in the second opening—and so on. From this we conclude that the effusion of blood does not take place in all parts at once. And as the interval of time between the appearance of the different jets is more considerable than it would be if

* When irritating only one nerve upon one side, it amounted to four times the quantity I obtained in the ordinary state. In other cases I got much more.

these differences depended upon the propagation of the nervous irritation, the opinion that the abundant effusion of blood almost exclusively takes place in the posterior bulb, and from this distributes itself into the venous cells, progressing from the posterior to the anterior part, is justified.

I have not yet referred to the corpora cavernosa penis. Do they in the dog also participate in erection? They certainly do, but not until after the corpus cavernosum urethræ has begun to fill. To convince ourselves of this fact we make openings in those parts. When no irritation of the nerve takes place, only some few drops of blood, or none at all, appear in the wound; but as soon as irritation begins, first the corpus cavernosum urethræ commences to fill, and afterwards a jet of blood makes its appearance in the opening of the corpus cavernosum penis. But at present I know no means to decide whether the swelling of these parts is dependent on the erection of the corpus cavernosum urethræ, or if it possesses its own proper apparatus for erection.

Up to this point of the inquiry my opinion is founded on facts, and I should like to stop here, but the mind is enticed to propose further questions as soon as one is solved. Thus I ask further, in which way or by what means the effusion of blood in the penis is produced. Two hypotheses present themselves. First, we may assume that by any muscular arrangement the area of the arteries is increased when the nerves are irritated, and that in consequence of it the flow of blood is free from the resistance the smaller area naturally imposes. Kölliker and Kohlrausch incline to this view, but I have reason to object to some points in their conclusions. Kölliker considers erection as a kind of motion quite analogous to the movements of the heart, assuming that the muscular fibres in the walls of the venous cells and the smaller arteries are continually in a contracted state, produced by nerve fibres taking their origin from sympathetic ganglia, and that at the time of erection the effects of the ganglia become annihilated by the action of the spinal fibres, in consequence of which the area of the before-mentioned vessels becomes increased. But it seems very strange that during the long periods in which no erection occurs as in children, old men, &c., neither the muscular fibres nor the nervous system should become fatigued! This objection does not apply to the heart, the contraction and dilatation of which alternate regularly. If in erection there be any active distension of the vessels, it must be effected in some other way. The second hypothesis consists in the belief that there might be (under the influence of the nervous system) any molecular

alteration in the walls of the vessels, especially the veins, in consequence of which they may become softened, and may lose a portion of their rigidity, so that they may be more easily distended by the pressure of the blood. I prefer, however, to leave this question for the present, in the hope that it may be cleared up by further investigations.

OF VERY FINE NERVE FIBRES RAMIFYING IN CERTAIN FIBROUS TISSUES AND OF TRUNKS AND PLEXUSES CONSISTING ENTIRELY OF VERY FINE NERVE FIBRES IN THE BLADDER OF THE FROG.

By Lionel S. Beale, M.B., F.R.S.

Plate I.

Researches upon which I have been long engaged, have convinced me that the ultimate nerve fibres are much finer and more abundantly distributed than is generally supposed, and that the active terminal branches of many nerves where they ramify abundantly in tissues, have been included in the so-called connective tissue. The terminal branches of all nerve fibres are so very fine, as not to be visible by magnifying powers in ordinary use, many in the frog being less than the 1–100,000th of an inch in diameter. In man and mammalia they are wider than this, but appear as faint granular, and too often scarcely visible lines. In the frog, although so fine, they are much more distinct, and being firmer, are much more easily studied than in mammalia.

All peripheral nerve fibres are connected with nuclei (germinal matter), but these nuclei are separated by much greater distances in the nerves distributed to some tissues than others. The nuclei are for the most part o v a l, but in some cases they are t r i a n g u l a r. These bodies, which exist in great number in many sensitive surfaces, as for example, just beneath the epithelium of the mucous membrane of the fauces, have been, and are still, considered by many authorities, to be connective tissue corpuscles. Not a few observers in this country, as well as on the continent, following Virchow and his school, consider that they communicate with each other by tubes, and thus form a new canalicular system for conveying nutrient juices.*

The nuclei a r e n e v e r t e r m i n a l but a fibre always

* See a paper on the distribution of the nerve fibres to the mucous membrane of the human epiglottis.—"Archives," vol. iii, page .

passes from each nucleus in two or three different directions. Both nuclei and fibres have by many observers been included under the head of connective tissue. Nor, indeed, can these fine terminal nerve fibres be demonstrated in fibrous tissues in which they exist in great number, by the ordinary processes employed in demonstration. They can only be seen in exceedingly thin sections and with the use of the highest powers. Not only are nerve fibres present in certain forms of connective tissue, but there are many fibrous tissues destitute of vessels to which nerves are distributed. In the cornea, the fibrous tissue about the pericardium, the pericardium itself, and the bundles of fibrous tissue in connection with the vessels and various organs in the abdominal cavity of the frog, nerve fibres are very numerous.

I have succeeded in demonstrating nerve fibres in connection with the vessels in so many tissues of the frog, and of certain mammalia, that I am strongly inclined to the opinion, that in vertebrate animals nerve fibres exist wherever vessels are present. These remarks apply not only to the smallest arteries and veins, but also to the capillaries.* Since the capillaries are devoid of muscular fibre cells, and do not possess contractile power, it is probable that these fine nerve fibres associated with the capillary vessels are afferent fibres.

In the papillæ of the frog's tongue, for example, besides the bundle of sensitive nerve fibres passing up the central part of the papillæ, there are very fine nerve fibres distributed to the vessels, and some fine fibres in the connective tissue external to the vessels. Similar fibres exist in the small papillæ, to which neither vessels nor dark-bordered nerve fibres are distributed. The nuclei connected with these fibres are about the 1–300th of an inch or more apart. Now these fibres are not ordinary fibres of connective tissue, for I have traced them into undoubted nerve fibres. Moreover, the fibres and nuclei are much more abundant in connection with some capillaries than others. They are very numerous upon the smallest vessels of the ciliary processes of the eye (ox) as well as upon those which are provided with muscular fibre cells, and many are to be found in the connective tissue upon the free border of the finest vessels. I consider that these branches are in part afferent or excitor and partly efferent or motor nerves of the vessels.

The fact of the presence of undoubted nerve fibres in tissues destitute of vessels, and deriving their nutriment from the

* See figures 5 and 9 in plate xxiii, Phil. Trans., 1860.

plasma permeating vessels situated, perhaps at some distance, is another strong argument in favour of the existence of afferent nerves, bearing to the vaso-motor branches, the same relation as the excitor fibres bear to the spinal motor nerves. Such fine nerve fibres are distributed to the cornea of all animals, and very fine fibres ramify upon different planes in the substance of the proper corneal tissue. From their distribution we are justified in assuming, that these fibres are not ordinary sensitive fibres, but are, nevertheless, concerned in transmitting impressions of some sort from periphery towards nervous centres, while in certain morbid states they are probably instrumental in transmitting impressions which produce the sensation of pain. Ordinarily these afferent and efferent fibres preside over the nutritive process, and it is easy to conceive how any alteration in the amount of nutrition passing to the tissue must influence through the nuclei and afferent fibres the ganglia from which the vaso-motor branches take their rise. Thus the calibre of the minute arteries may be altered by the slightest modification in the supply of pabulum to the tissues outside capillary vessels, dependent upon any mechanical or chemical alteration in the tissue whereby the activity of the nutritive changes becomes altered. Normally, the balance between the quantity of pabulum taken up by the tissue, and that escaping from the capillaries would be maintained through these afferent and efferent fibres, and it is easy to understand how any derangement of afferent fibres, nerve centre, or efferent branches would disturb the nutritive process.

I think it certain that the rapidity of growth of tissues is determined solely by the supply of pabulum, and this supply is regulated and equalised by a special system of nerves which is however connected with the cerebro-spinal system, and may influence it, or be influenced by it. I have been led to the conclusion that nerves invariably form complete circuits, and that there are afferent or excitor nerves and efferent or motor nerves presiding over the nutritive processes, which may act independently of the cerebro-spinal nerves or centres. It will be asked, if I hold that there is a complete circuit in the case of the afferent, and another in that of the efferent fibres distributed respectively to the tissues and small arteries, or that the afferent and efferent fibres form part of the same circuit, in which case an impression might be transmitted to, and a motor impulse start from, the same ganglion cell. To consider this question would, however, require many pages, and I must, therefore, postpone it for a separate communication.

The fine nucleated fibres I have described distributed in the neighbourhood of capillary vessels, and to tissues, which do not receive a vascular supply at all, form in the tissues of the frog generally, fine trunks consisting of several very fine fibres, and these unite to form larger trunks which, as a general rule, are accompanied by one or more dark-bordered fibres, but in the bladder, in the heart, and also in the mesentery, large trunks exist which are composed entirely of these very fine fibres, and at certain points plexuses are formed. In Plate I., one of these plexuses with fibres passing from it in different directions, from the bladder of the frog, is represented. In the cornea the individual fibres are not so distinct, nor are the fibres so decidedly separated from each other as in the drawing. Many seem to be in course of splitting, an appearance more like that seen in the sympathetic branches of birds and mammalia, where the fibres in a trunk appear to be connected together forming bands.*

It is quite certain, therefore, that these fine fibres are independent of the dark-bordered fibres. But, it will be asked, are all the fine fibres in the trunks—for example in those represented in the figure—afferent fibres? In a trunk passing from the cornea, doubtless, all are of this nature, but I have seen many such fibres passing amongst the muscular fibre-cells of the bladder and also to the contractile coats of the small arteries, so that at least in this case it is probable that some of the fibres entering into the formation of the plexus in Plate I., are afferent and others efferent. There are no characters by which one class of fibres can be distinguished from the other. Amongst the nerves forming the large bundle which supplies a limb, some bundles of fine fibres which probably belong to the same class are to be found, but I have never seen large bundles of very fine fibres like those in the bladder and mesentery, in the voluntary muscles. Such bundles, however, do exist in connection with the heart.

The bundles of fine fibres at their peripheral distribution form plexuses and networks. I have never seen any termination in any case. The fine nerve fibres distributed to small arteries and veins also form networks, and very fine fibres can be traced ramifying amongst the muscular fibre-cells on different planes. Kölliker suggests, that such fibres ramifying on the outer part of small arteries and veins distributed to voluntary muscle, and on fine vessels on the arterial side of

* The arrangement of the nerve fibres in the cornea of various animals is fully described in an elaborate paper by my friend and former pupil, Dr. Ciaccio, of Naples.

capillaries destitute of a muscular coat, are of the sentient kind. The latter fibres, as I have stated, are probably afferent or sentient, but his remarks on this question are very undecided, and he does not profess to have studied the subject carefully. It is very hard to conceive what purpose could be served by the free distribution of sentient fibres upon and in the substance of the muscular coat of an artery. Some of the fibres running with vessels distributed to voluntary muscles are motor branches, which, after running parallel with vessels for some distance, diverge and are distributed to the muscular fibres. Kölliker considers certain nerves for the most part on the surface of the muscle as sentient fibres, but he adduces no facts to show that this view is correct.*

It is important that I should state definitely that the bundles of very fine fibres, distributed to the frog's bladder and in other tissues, are not visible in specimens prepared in the ordinary manner and examined in water or weak glycerine. In the bladder from which the specimen, figured in Plate I., was taken, there was no appearance whatever of these very fine fibres when the specimen was first prepared, but after the prolonged action of dilute acetic acid, a great number of bundles, many of which were as much as 1-2,000th of an inch in diameter, and very many finer compound fibres, made their appearance. The vast majority of these bundles of fine fibres were not only destitute of true dark-bordered fibres, but of any one fibre more than the 1-60,000th of an inch in diameter.

It is scarcely probable that any observer will doubt that the fibres, figured in Plate I., are true nerve fibres. Their mode of arrangement, the manner in which the trunks branch and ramify amongst the muscular fibre-cells, the character of the nuclei connected with the fibres, and the change produced in them by the action of acetic acid, show them to be nerve fibres. I have already proved that very fine fibres form the continuation of dark-bordered fibres, and that fibres, as fine as some of the finest of these, ramify in the same sheath with the dark-bordered fibre, even in the case of the dark-bordered fibres distributed to voluntary muscle (Phil. Trans. 1862).

But that these very fine fibres in the bladder, which I believe have now been demonstrated for the first time in my specimens, are true nerve fibres is placed beyond all question by the fact of their being continuous with ganglion-cells. I have seen several ganglion-cells from which such fine fibres alone (every one being less than the 1-50,000th of an inch) proceed.

* Croonian lecture, Proceedings of the Royal Society, 1862.

From different parts of one ganglion-cell sometimes six or seven or more very fine fibres may be traced, while not a single dark-bordered fibre comes near to the cell or bundle of fibres under consideration.

I fear the accuracy of these observations may be questioned by many fellow-workers in Germany, and more especially by those of the Dorpat school, and the difficulty of preparing the specimens is so great, that my conclusions are scarcely likely to be confirmed for some time to come. The appearances are, however, so distinct that I have been able to demonstrate the most important points to my class; and, as my specimens will keep for a considerable length of time, they can be examined by any one desirous of seeing them.

It would seem then that in the frog these fine fibres are distributed to capillary vessels, to fibrous tissues devoid of capillaries, to the tongue and palate, to the unstriped muscle of the bladder, pharynx, gullet, stomach, and intestines, to the unstriped muscle distributed to the coats of arteries, and to the muscular fibres of the heart, and probably they are to be made out in many other tissues than those above named.

I am unable to enter fully into the question of the distribution of the different classes of nerve fibres to the various tissues of the frog's bladder, nor can I discuss satisfactorily their several offices; but on these important questions I would offer the following remarks :—

With reference to the kind of nerve fibres, it is certain that—

1. Dark-bordered fibres are distributed to the bladder of the frog, and that the very fine terminal fibres, resulting from the subdivision of these, are freely distributed with the ultimate branches of other nerve fibres.

2. That there are fine fibres running in the same sheath with the dark-bordered nerve fibres, as I have described in the case of dark-bordered fibres distributed exclusively to voluntary muscle. See Archives, vol. III., and Phil. Trans., 1862 (just published).

3. That there are very many bundles of very fine fibres which sometimes run with dark-bordered fibres and sometimes also form special trunks destitute of dark-bordered fibres (Plate I.).

4. That many of these very fine fibres are directly connected with ganglion-cells upon the outer surface of the bladder.

5. It is certain that many ganglion-cells have no dark-

bordered fibres whatever in connection with them; but I cannot feel confident that all the ganglion-cells are unconnected with dark-bordered fibres.

In considering the function of this most elaborate and beautiful nervous arrangement, it must be borne in mind—

1. That the muscular fibre-cells and vessels of the bladder are freely supplied with nerves.

2. That nerves ramify upon the surface of the mucous membrane.

3. That the bladder contracts when the nerve-fibres, distributed to the skin of the animal, are irritated, and its contraction seems also to be under the influence of the will of the animal.

Now, I think it probable that the nerves, distributed to the muscular fibre-cells of the bladder, are branches of the same trunks as those distributed to the vessels, and are connected with the ganglion-cells. There can be little doubt that some of these fibres are afferent and others efferent, and that the contraction of the muscular fibre-cells is effected through the influence of these fibres. As already stated, I conclude that the numerous nerve-fibres in the cornea and other fibrous tissues are purely afferent, and that they indirectly influence the motor fibres distributed to the nearest vessels. In the bladder there are afferent fibres corresponding to these, and efferent or motor fibres distributed to the vessels, and also to the muscular fibre-cells.

Whether the dark-bordered fibres are purely sensitive, or whether some spinal motor fibres thus pass directly to the bladder, I am unable to say. It is probable that the fine fibres running with the dark-bordered fibres of the bladder (Archives, vol. III., Plate XVII.), correspond to those in the same sheath with purely motor or sensitive dark-bordered fibres. It is, however, not possible to discuss this question advantageously until many points in connection with the general distribution and function of the different classes of nerve-fibres are cleared up. I shall have to consider this question more fully in a future communication, and must for some time to come attempt little more than to describe the arrangement of the nerve fibres I have demonstrated.

A CASE OF RAPID "WASTING PALSY" FROM STRUCTURAL DISEASE OF THE SPINAL CORD.

By J. L. W. Thudichum, M.D., Lecturer on Chemistry.

THE INVESTIGATION OF THE NERVOUS CENTRES WITH COMMENTS.

By J. Lockhart Clarke, F.R.S., &c.

Case.—A gentleman, æt. 54, had enjoyed good health during adolescence and early manhood; when at the age of 45, he met with what was then considered a slight accident. In jumping across a flower-bed for a wager, he came down heavily on his heels, and then fell backwards upon his head. He was stunned for a time, but gradually recovered, and after some days' confinement to his bed, appeared to be quite well again.

It was, however, soon perceived that a great change took place in his habits. Having been extremely fond of manly sports and exercises, rowing, cricketing, riding on horseback, dancing, and the like, he discontinued to take part in any of them, although he continued to go every autumn to the Scotch moors for the purpose of shooting grouse.

At the age of 50, while engaged in this latter sport, he perceived that his right leg had lost a part of its usual strength; that although he could perform with it every motion, yet he could not perform it in the manner necessary for the purpose; that he could not put the foot into the exact place where he wanted to put it. The weakness of the limb increased, and the right arm began to show similar symptoms. Under these circumstances, various remedies were tried in vain, until acupuncture and the electrical induction current, effected a speedy recovery.

Four years afterwards, however, the symptoms of weakness in the leg and arm re-appeared, and were not checked by the remedy which apparently had arrested and cured them before. Notwithstanding the treatment of the most celebrated physicians in town and country, the affection terminated in complete motor paralysis of the right leg. Now the left leg began to be effected in the same manner as the right; the arms, though weakened, retaining sufficient power to enable the patient to walk with the assistance of two sticks. While the left leg became quite paralysed, the right arm exhibited the same symptoms of weakness, lost all power in the

most gradual manner, and became ultimately quite paralysed. This was succeeded by gradual paralysis of the left arm. There remained some motility in the toes of both feet, and a slight amount of flexibility in the left index finger, as also slight adducting power in the left thumb. Sensibility was everywhere perfect and nowhere notably increased.

While the limbs thus lost their motor power, they gradually wasted in the most remarkable manner. The calves became two flat planes, what muscular substance remained upon the gastrocnemei being at the side; the thighs shrank very much; the arms and hands were wasted to a very extraordinary degree, the right hand particularly appearing as if crushed, having lost the whole of the projecting mass of the short muscles of the thumb.

In the right hand there had at one time been some involuntary movements, but they had entirely ceased; in the index finger of the left hand, which retained a small amount of voluntary motion, this spasmodic, but painless motion, came at intervals to the last. The hand lying queitly by the side of the patient, the fingers, stretched out, moved up and down, more or less rapidly, like that of a person striking a key of a note on the pianoforte. This was the only spasm of a clonic nature observed throughout the whole illness.

The only places where there was pain, for any length of time, was at the points of insertion of the deltoid muscles. At the insertion of the right deltoid, in particular, a hardness could be felt, which was painful to the touch. Those muscles were much wasted, so that the shoulders were angular, with projecting points of bone, and flat planes corresponding to the places covered by the deltoid muscles.

While this wasting palsy gradually crept over the patient, various modes of treatment were adopted, according to the opinion of the one or the other of the physicians consulted on the case. The ordinary medical attendant prescribed alterative doses of bichloride of mercury, subsequently iodide of potassium; steel medicines, quinine and others were tried in vain; acupuncture and all forms of electricity failed to have any effect. The seventh cervical vertebra was very prominent, and appeared to the touch as if thickened; indeed, so deceptive was the condition of that part of the spine, that several of the gentlemen consulted, had diagnosed a tumour in that region, which was supposed to cause the paralysis, by pressure upon the spinal marrow. Cupping had been repeatedly applied to this prominence, blisters had been raised, and ointments lavished upon it; but neither tumour nor paralysis had been affected thereby. There was no acquired

constitutional taint of any kind that could be ascertained; the surface of the skin was everywhere clear and white.

The senses and all mental functions were perfect; but a state of sleeplessness gradually developed itself, which ultimately constituted the most painful, and least remediable, symptom of the whole disorder. The patient was always very warm, down to the most extreme parts, and could under no circumstances bear to be too warmly covered. He had frequent attacks of what may be called showers of heat, that is, sudden and rapidly passing sensations of heat all over his body; there were no rigors or goose-skin. The pulse was sometimes natural, always full, mostly accelerated to 100, 112, and even more beats per minute.

Gradually the muscles of the trunk participated in the palsy, less in the wasting. The first effect of this extension of the disease, was inability to sit in the erect posture, next came inability to strain at stool or while making water. The contractility of the bladder was unimpaired, and the urine was expelled regularly and completely, when time was given for the operation.

It soon became necessary to assist defæcation by the injection of water and the administration of purgatives. Even then the sphincter, which was not paralysed, but strongly contracted, and contractile at will, would cause retention of the solid part of the faeces, and allow only the water and a small amount of soft matter to escape. This difficulty at first was obviated by artificial compression of the abdomen, by means of an air pad and leather strap, which was tightened as often as the patient strained, and immediately loosened again. Notwithstanding these precautions, an accumulation took place in the greatly dilated rectum. An accumulation of flatus in the large intestine, was relieved by passing a long tube into the bowel; the pain round the rectum persisting, and retention of urine taking place, the rectum was digitally explored, and the accumulation, with a clear passage through its central part was discovered. The faeces were broken down with two fingers, while a current of warm water was passed through the rectum, and by this operation, which was somewhat difficult, the pain, retention of urine, and obstruction of the bowels, were removed. Shortly after, a tetanic spasm of the recti muscles of the abdomen set in, which was painful, and lasted for about 14 hours. It was removed by small doses of prussic acid and morphia, and the application to the abdomen of hot flannels.

The most distressing symptom during the last six weeks of the illness was catarrh of the lungs, which caused a slight

amount of bronchial secretion. The phlegm proceeded to the trachea, and remained there, as the respiratory muscles were paralysed, and coughing was impossible. By slight movements of the diaphragm, which was not paralysed, and was the organ, which in union with the respiratory muscles of the neck, particularly the stero-cleido-mostoidei, performed ordinary respiration; the lump of phlegm in the trachea could be moved up to the larynx, but it could not be carried through the rima glottidis, and there produced the most anxious sensation of suffocation. When I was first called to the patient about this symptom, he had been struggling with a lump for three hours. I immediately performed a kind of artificial coughing by assisting the patient's feeble efforts with a synchronous pressure of both hands upon the lower part of the thorax, the servant compressing the abdomen steadily with both hands. The phlegm was immediately carried through the larynx, and the patient was relieved. The attendants were taught the manner of practising this operation, and during the last three weeks of the patient's life it had to be frequently performed.

Quinine, morphine, strychnine, iron, phosphoric acid, and a variety of other remedies had no effect upon the disease, which from first to last, within a period of nine months, progressed to a fatal issue, without a halt, change, or hesitation. Morphine produced some sleep, but left an irritability, which caused the patient to wake up in great fright; sometimes mucus in the throat was the cause of this symptom. The mercurial and iodide remedies had left, if any, no beneficial effects; and the iodide, which had been continued upon the advice of a physician, with the hope of its reducing the tumor in the back of the neck, had probably had a great share in the production of the condition of the air passages. In the earlier stages the patient had drunk much wine—less latterly; but when faintness came on, after setting up for defaecation, or after shifting from the bed upon his chair, he usually took some. Against brandy and the like he used to protest strongly, as it left him in a state of great heat with excitement of the circulation. Upon my advice he confined himself to claret, and brandy was reserved for those rare occasions, when strong stimulants were medically indicated. He underwent the process of the Turkish bath on repeated occasions, and felt revived by it; but as the only bath at his disposal unfortunately broke down at the time when it might perhaps have been useful, it cannot be said that this agent had any trial in this case. It was perceived that the limbs of the right side, which had been paralysed the longest, and retained the least motility, perspired the most readily.

He died of a very gradual cessation of the respiratory powers. The heat continued strong to the last, and the pulse could yet be felt, after respiration had ceased, and consciousness had fled. During the last hours there had been many attacks of heat showers and copious perspirations.

Post-mortem examination eighteen hours after death.—There was the usual lividity and discoloration of the skin, especially about the legs. The wasting was greater on the right side than upon the left. The right hand was flattened, as if crushed. On the back was seen a prominent tumefaction involving the seventh cervical and two first dorsal vertebræ. The spinal processes were distinguishable, but there was a thickening on either side, so that the finger could not be pressed down there. The third dorsal vertebra was natural. The tumefaction was hard, dense, and could not be compressed. The skin showed numerous scars of cupping wounds, and the puckering and discoloration left by blisters. When the skin was removed the tumour was seen to be a layer of hard fat, of the size of a crown piece, half an inch thick in the middle, and gradually getting thin towards the circumference. The spinous processes, and all tissues around, were found in a perfectly normal condition.

The spinal canal was next opened; it was nowhere changed, with the exception of a small place on the fifth dorsal vertebra, where there existed a somewhat varicose vein. The position and appearance of the chord were normal. It was carefully removed and given to Mr. Lockhart Clarke for examination. The brain showed nothing abnormal. On the declivity of the basilar bone, however, immediately behind the back of the Sella Turcica, there were two bony excrescences, covered in by fibrous adhesions of the dura mater, but when isolated, appearing as sharp, spinous projections. The left one was situated somewhat higher, and projected an eighth of an inch from the surface of the bone. The right one was situated a little lower, and was a sharp conical spine, three-eighths of an inch in height. There were irregularities in the basilar bone, resulting in a protrusion of the margin of the foramen magnum upwards, and the narrowing of the apertures for nerves and blood-vessels. But it was difficult to ascribe to these irregularities any particular origin or effect, as they were found in most skulls examined for the purpose of comparison. The spinous excrescences if they affected the brain substance must have irritated the pons Varolii; no permanent impressions could, however, be found on this part. If these excrescences and irregularities could be considered as the effect of injury, it is perhaps right to bring them in connection with the fall ex-

perienced by the patient nine years before his death. The absence of brain symptoms speaks against the hypothesis that these excrescences had anything to do with the disease of the chord of which the patient died.

The chest and abdomen were not opened. The part of the right deltoid muscle, which had been painful during life, was dissected, but no changes were discovered in it. Various parts of muscle were examined and compared. The paralysed muscles remained of a pale rosy hue when exposed to the air, while the muscles which had been contractile became dark red as usual. Examined with the microscope the paralysed muscle exhibited the fibrils in a state of relaxation. While in the healthy muscle the fibrils are closely folded together, so as to produce the tranverse striæ, in the paralysed muscle all tranverse striæ had disappeared, and the fibrils lay in undulating lines. Beyond that, there was no change even in the most wasted muscle. There was no fatty or other deposit of any kind, either in or upon the microscopical elements.

The fatty bolster on the back of the neck consisted of common fat tissue, with very little connective tissue.

INVESTIGATION OF THE NERVOUS CENTRES, WITH COMMENTS.

By J. Lockhart Clarke, F.R.S.

(1) The parts which I received from Dr. Thudichum for examination consisted of the cerebellum and pons varolii, the medulla oblongata, and the whole of the spinal cord. They had been carefully removed soon after death, and were all free from mechanical injury. Upon external examination the cerebellum, pons varolii, and medulla oblongata presented no evidence of disease; but in the white columns of the cord there was observed a variable extent and degree of softening, which was greatest at the upper part of the lumbar and the lower part of the dorsal regions, where it was confined chiefly to the posterior columns, and some parts of the lateral, as will be described in proper order. At these places the cord was divided transversely by a very sharp instrument, and small portions of both the white and grey substances were at once examined under a $\frac{1}{8}$th-inch object-glass, but without detecting, among the confused mass of broken nerve-fibres, granules and nerve-cells, any satisfactory evidence of morbid product.

The mode of preparing these parts for minute examination

was similar to that which I employed for the same purpose on former occasions.*

(2) I began the examination of the cord at its lower end, and shall begin from the same part to describe the extensive and remarkable morbid appearances which render this case so peculiarly interesting and important. The central part, or axis, of the filum termile was occupied by a comparatively thick column of dark, morbid substance, which entirely obliterated the canal and replaced the small remnant of grey substance by which, in the normal state, it is here immediately surrounded. In very thin sections—which, however, from the friability of the substance were not easily made—the structure of this morbid material could be ascertained under a high power without disturbance, and by this means was found to consist of a dense aggregation of cell-like bodies, intermixed with corpora amylacea and a few fine fibres. The cell-like bodies, fig. 1, A, Plate II., were more or less globular, and varied from about the 1300th to the 2800th of an inch in diameter. Their outline was delicate, and in many instances resembled that of scaly epithelium, or of certain vegetable cells. I believe, however, that they were not real cells, but consisted partly of, more or less globular portions of the white substance of diseased or degenerate nerve-fibres. At *a*, fig. 1, the coarsely granular substance was clearly seen, in many instances, to result from the breaking up of these cell-like bodies. Of the epithelium which originally surrounded the canal no traces could be distinguished, except, perhaps, the fine fibres which once proceeded from their tapering ends, and now intersected the mass. The corpora amylacea were not particularly abundant; but in two or three places the dark opaque mass was interspersed with small patches of granular degeneration.

(3) In ascending to the lower part of the conus medullaris, the diameter of this morbid column increased nearly in the same ratio as that of the cord, obliterating the canal and replacing nearly the whole of the grey substance, as represented in a transverse section, fig. 2. To appreciate the exact extent of the morbid changes which were thus induced, it will be necessary to compare this section with one taken from the corresponding part of a healthy cord, as shown in fig. 3. By this comparison it will be seen that the morbid cylinder (*a a*) encroached forward on the deep central portions of the anterior white columns (*e, e*), which are consequently shallower than in the healthy section; and that it destroyed and replaced the whole of the grey sub-

* See Beale's Archives of Medicine, No. 9; and British and Foreign Med.-Chirurg. Review, July, 1862.

stance except the superficial part in front and at the side (*d*), and the gelatinous substance behind (*b*); from both of which several slender processes, consisting chiefly of connective tissue, intersected, and divided it into lobes of different sizes.

Through the rest of the conus medullaris the morbid column increased somewhat in thickness, and assumed an oval shape, with its long diameter behind-forward. On comparing a transverse section of the white and grey substance (fig. 4) with a similar section from the corresponding part of a healthy cord (Fig. 5), the difference observed is very striking. The relative quantity of grey substance destroyed is less than in fig. 2; but the morbid column (*a*) has obliterated nearly all of that portion which immediately surrounds the canal, including the inner sides of the posterior cornua, which seem to be tilted backward, as it were, by the forward pressure of the morbid column against the anterior commissure and diminished white columns (*e, e*). The chief portions, however, of the anterior cornua (*d, d*) still remained, and with one exception, to be described in its proper order, were found healthy.

(4) Through the lower part of the lumbar enlargement the morbid column continued to increase somewhat in size, but chiefly in an antero-posterior direction, and the changes which it effected in the form of both the grey and white substances were remarkably curious, as may be seen by comparing together corresponding sections of the diseased and healthy cord (figs. 6 and 7). In fig. 6 the posterior white columns (*c, c*), although scarcely, or not at all, diminished in area, are very much displaced and altered in shape by the presence of the morbid column (*a*), which seems to have been developed between them, from the bottom of the median fissure, to be the result of diseased blood-vessels, as we shall see further on. Thus originating and continuing to increase in size, the morbid mass pressed forward the transverse portion of the grey substance with the central canal, while in the same proportion the anterior white columns (*e, e*) were reduced in depth. As effects of the same cause, the posterior cornua were not only separated from each other to an unnatural extent, but were increased considerably in length; while the anterior cornua (*d, d*), on the contrary, were shortened to corresponding degree.

The central canal was not entirely obliterated, but was less distinct than usual; while in front of it, in the substance of the anterior commissure, was a transparent and elongated patch of granular degeneration (*h*). The roots of the nerves, especially the posterior, were, as in the previous sections, reduced considerably in thickness.

On ascending through the lower half of the lumbar enlargement, the morbid column (*a*) gradually diminished in size, until, a little below the middle, it entirely disappeared. In the process of reduction the mass diminished laterally and from behind-forward, so that its anterior portion was the last to disappear in the form of a longitudinal cone or tapering cylinder resting on the posterior commissure between the bases of the horns. In the same region of the cord the proper substance or healthy portion of the posterior columns, especially the deeper parts, was affected with softening, which, however, disappeared gradually towards the middle of the lumbar enlargement, where, for a short distance, it was limited chiefly to the surface. At this point the grey substance was not, either in form or size, appreciably altered from the natural state; and with the exception of a peculiar condition of the nerve-cells, to be noticed hereafter, had nearly a healthy appearance. Above this point, for about half an inch in the middle of the lumbar enlargement, the area of the softening in the posterior columns again increased—their superficial halves, between and immediately behind the heads of the cornua, being quite pulpy and disorganised. In this destructive process a large proportion of the fibres of the posterior roots of the nerves were of course involved.* On the right side of the cord the posterior part of the lateral column, extending from the posterior-lateral fissure to the base of the caput cornu, and comprising a triangular area, was also softened to a very considerable degree.

Through the rest of the middle of the lumbar enlargement, the softened state of the posterior columns diminished in area, but not in degree, and was gradually more limited to the surface.

(5) Throughout the upper third of the lumbar enlargement the whole of the posterior columns was again involved in the softening. In degree, however, this morbid condition was greatest at their deeper parts (fig. 8 *c'* Plate III), where they rest on the transverse commissure of the grey substance, which in this case, as shown in the figure below *c'*, was eroded, as it were, and granular. In some sections as represented in fig. 8, this more particularly softened portion of the posterior columns was separated from that which was firmer and more super-

* Perhaps it may be well to remind the reader that the posterior roots of the spinal nerves are attached *exclusively* to the *posterior* white columns, and that none of their fibres are, as they were formerly supposed to be, attached immediately to the lateral columns. These facts I first pointed out in 1851. Phil. Trans.

ficial by a process of pia mater or connective tissue, which extended from the posterior vesicular column (*k*) on the left side to caput cornu and posterior column on the right. Fig. 9 represents a transverse section of the grey substance at about the middle of the upper third of the lumbar enlargement.* Here we find that the posterior vesicular columns (*k, k*), although not much reduced in size, are somewhat altered in position and shape. Along the outer side of the right vesicular column was a curved tract of granular degeneration, extending backward to the base of the caput cornu. The anterior cornua, *d, d*, were somewhat reduced in size and unsymmetrical in shape; and the anterior median fissure (*s*), instead of being midway between them, and equally dividing the anterior white columns (*e, e*), extended from the right extremity of the transverse commissure along the side of the corresponding cornu. Within the fissure was an enlarged and apparently unhealthy blood-vessel, reaching backward as far as the commencement of the tract of granular degeneration, of which it seemed to have been the cause. Fig. 10 represents another section from the same region, a little higher up. Here the form of grey substance on both sides is strangely altered. On the right side the posterior vesicular column (*k*) is not greatly reduced in size, although very much modified in shape. On its outer side in the substance of the cervix cornu posterioris, is a pyriform mass of finely-granular degeneration (*m*). This morbid space, as well as that in the previous section, presented, under a high magnifying power, precisely the same appearance as did those which I described in my former cases.† On the left side of the section, the posterior vesicular column had entirely disappeared: not a cell remained; while the cervix cornu, (*n*) in which it should have been enclosed, was extraordinarily narrow, and gave off numerous processes of connective tissue, which, toward the mesial line, interlaced and became continuous across the posterior columns (*c, c*), with similar processes from the opposite side. Fig. 11 represents another curious deviation from the natural form of the grey substance at the upper end of the lumbar enlargement. The abnormal appearance will be at once evident on comparing it with fig. 12, which exhibits the right side and transverse commissure of the grey substance from the corresponding region of a healthy cord. In fig. 11 the posterior vesicular column (*k*), on the left side is partly thrown or drawn

* Compare this with fig. 13, plate xxi, of my "Researches on the Grey Substance of the Spinal Cord." Phil. Trans., 1859.

† Beale's Archives, No. 9, 185; and British and Foreign Review, July, 1862.

across the middle line, and united to its fellow on the right side by a broad process of connective tissue which separates the posterior white columns (*c c'*) between them, into two separate ovoid masses; while the transverse commissure (*p*), by an increased development of connective tissue, is much broader than in health. Moreover, the entire lateral halves of the grey substance have approximated, or been drawn more closely together.

Throughout the whole of this region (the upper third of the lumbar enlargement), all the white columns were more or less softened. The degree of softening was greatest in the posterior—particularly on the right side, at their deeper parts, where they rest on the transverse commissure, which, in many sections, was also in the same condition, and more granular than usual. At this latter part of the posterior columns there was generally an oval or circular space in which they were quite pulpy. Next in degree of softening were the anterior white columns; and lastly, the lateral. In this region the cord, although most carefully hardened, was so exceedingly friable through its whole thickness, that the greatest difficulty was found, even with the sharpest instrument, in making sections sufficiently thin and perfect to obtain a correct view of the actual condition of the grey substance. Nevertheless, I may guarantee that all the figures that illustrate this case are exact delineations of the morbid appearances in the form in which they really existed.*

(6) At the lowest part of the dorsal region, where it gradually swells into the upper part of the lumbar, all the white columns were somewhat softer and more friable than natural, the posterior in particular, although they were less so than in the region last described. But the most remarkable and curious feature at this part of the cord was the form of the grey substance. Fig. 14, Plate IV., represents its appearance in a perfectly healthy cord. Here we find the lateral halves of the grey substance, with their posterior vesicular columns (*k, k*), standing widely apart, and joined only by the narrow transverse commissure (*p*). But in the morbid section, fig. 13, the two lateral halves are drawn closely together, so that the posterior vesicular columns (*k, k*) are nearly in contact in the middle line; and while the gelatinous substance of the opposite sides is almost continuous behind them, the transverse commissure and space (*p*) around the canal have assumed a triangular form. An

* Some of these figures with the preparations from which they were drawn were exhibited by Dr. Thudichum at one of the meetings (December 8, 1862) of the Medical Society of London.

approach to this arrangement is seen in fig. 11. Now what renders this form of the grey substance so exceedingly curious, is the fact that it bears a striking resemblance to the natural form of the same substance in the dorsal region of most of the mammalia, as may be seen on comparing my figures in Phil. Trans. 1851 and 1859, and in Beale's Archiv. No. 3, vol. iii. The tractus intermedio-lateralis (*t*) was very distinct and prominent.

Through the upper half of the lower third of the dorsal region, the whole of the posterior columns were very soft. The posterior parts of the lateral columns also were softer than natural, while the grey substance was still much altered in shape. Fig. 15 represents a transverse section of this substance at the upper part of this region. (*c c'*) are two remaining portions of the posterior columns between the lateral halves of the grey substance. Behind (*c*) the posterior cornua are united by a process (*a*) of connective tissue; and between it and the other (*c'*) were several morbid patches (*b, b, b*), which, when seen under a low power by reflected light, appeared nearly black, but by transmitted light, were exceedingly transparent, and under a high power were found to be finely granular degenerations. These patches extended only a short distance upward, for after two or three thin sections, they entirely disappeared. Immediately above this point, that is, at the lower part of the middle third of the dorsal region, the posterior and lateral columns became much firmer, but after a few more sections, their deeper portions between the roots of the posterior cornua were again softened, particularly on the left side. Through the same space and the rest of the middle third of the back, the form of the grey substance was variously abnormal, the two sides being frequently unsymmetrical, while many of its blood-vessels were much enlarged. Fig. 16 represents a transverse section.

(7) At the beginning of the upper third of the dorsal region, the most important morbid condition was found on the right side in the substance of the anterior cornu, and immediately in front and on the outer side of the posterior vesicular column (*k*, fig. 17). This consisted of several clots of blood (*b, b*) effused from an adjacent vessel, which had a somewhat varicose appearance, and seemed partially eroded as if from granular degeneration. In the next section, fig. 18, which is much more highly magnified (60 diameters) a large irregular clot (*b, b, b*) was found in front of the posterior vesicular column (*k*), and therefore in the centre of the grey substance of the right side. Laterally it projected into the tractus intermedio-lateralis (*t*). The posterior white columns, especially

their deeper portions, overlying the transverse commissure, were softened to a considerable degree. These appearances extended for about the third of an inch up the cord, gradually disappearing; but beyond this point, the vessels in the neighbourhood, and frequently in the white columns, were larger than natural, and sometimes irregularly dilated; while the grey substance was curiously altered in shape and symmetry, as shown in fig. 19, Plate V. Here the anterior cornu (*d*) on the left side is smaller than that on the right, and is thrown or drawn backward away from the anterior median fissure (*s*), together with the corresponding tractus intermedio-lateralis (*t*), posterior cornu, and the posterior vesicular column (*k*), which is narrower and longer than its fellow. Within the caput cornu posterioris, or the extremity of the horn, on the left side, the blood-vessels were very much enlarged,—so much so, as to present a very unnatural appearance, even under a low magnifying power. In different parts of the grey substance, also, along this region of the cord, the blood-vessels were not only dilated, but accompanied by either roundish or irregular patches of extravasated blood.

The same condition in a greater or less degree, continued as far as the part where the cord begins to swell into the cervical enlargement. Here, on the left side, several very much dilated blood-vessels were observed. In some sections three, in others four, were found at the base of the tractus intermedio-lateralis, at its junction with the posterior cornu; and two or three others were seen at the outer part of the anterior cornu. Around the larger of those in the former instance, there was, in some sections, a granular transparent space containing many extravasated blood corpuscles more or less altered in appearance. The posterior white columns—particularly their deeper portions—were very soft; and the posterior part of the lateral columns also were softer than natural.

(8) On proceeding further upward through the lower part of the cervical enlargement, three or four dilated blood-vessels made their appearance in the centre of the grey substance around the canal (see fig. 20, *b*, *b*). After a few sections, two or more of these blood-vessels were seen to be surrounded by finely granular and transparent substance (*b*, fig. 21), which gradually increased in area for about half an inch upward, while the blood-vessels themselves were partially destroyed, evidently by granular degeneration. Fig. 22 (*b'*) represents this morbid area.* It will be observed, also, that the two halves of

* For the economy of space the ends of the posterior horns are not represented in the succeeding figures.

the grey substance are unsymmetrical. The posterior columns generally were much firmer, except at their deeper parts (*c'*) where they were soft and granular, and continuous with the morbid area, (*b'*) so that the transverse commissure was involved in the disease.

This condition of the white and grey substance gradually increased as it ascended the lower half of the cervical enlargement. The softening of the posterior white columns extended toward the surface, until it involved their entire area. Figs. 23 and 24 (*b, b'*), show the transparent areas of granular degeneration in two different sections. At the part from which the lower roots of the third dorsal nerves take their origin, this morbid space diminished, and rather suddenly disappeared, only two or three small transparent spots of degeneration remaining at the bases of the posterior cornua. The posterior white columns were somewhat soft.

Opposite the middle roots of the third dorsal nerves, the granular degeneration again made its appearance as a large transparent triangular space, which destroyed the whole of the posterior, and nearly all of the anterior commissure (see figs. 25 and 26 *b*). In fig. 26 the dark granular mass (*a*) is what remains of the fibres of the anterior commissure. It is separated from the anterior columns (*e, e*) by a layer of the soft and transparent granular substance (*b'*). Through each side of this morbid space, ran a branch of the blood-vessel which enters at the anterior median fissure. These branches at some points were partially involved in the degeneration, or seemed to be irregularly eroded. These morbid states diminished somewhat through the upper parts of the same region, but without entirely disappearing. The form of the grey substance was normal. The posterior columns were less soft, except at their deeper parts, which were quite pulpy.

(9) On a level with the lower roots of the second dorsal nerves, the degeneration again increased in extent, and destroyed the whole of the anterior and posterior commissures; but after extending upward a few lines, once more diminished. Fig. 27 (*b, b'*), Plate VI., represents its exact form and size at the middle of the part from which these nerves arise. At (*b*), the posterior commissure and parts surrounding the canal were quite broken up, consisting of bits of altered nerve-fibres, of granules, and of corpora amylacea in abundance. On the left side, the morbid area (*b'*) was larger than on the right, and completely destroyed the posterior vesicular column, which in this region consists of much smaller cells. Into this part of the morbid space the ends of some blood-vessels, completely thinned away as if

by erosion, were seen to project. The posterior white columns were soft only at their deep parts which overlay the transverse commissure. The other columns were apparently sound. On a level with the upper roots of the same (second pair of dorsal) nerves, the transparent area of degeneration was prolonged backward on the right side, as well as on the left; and for a distance of a few lines up the cord the whole inner portion of each cervix cornu, (or the parts between the transverse commissure and the caput cornu) were destroyed by irregular patches of granular degeneration, of different shapes and sizes, which presented a very striking resemblance to the holes in a moth-eaten cloth, as represented in fig. 28 (*b b′ b″ b‴*), &c. The spaces, however, were not vacant, but filled with a finely granular and transparent substance,—the granules, as I found in former cases, being mostly toward their centres. On the left side these morbid appearances gradually diminished on ascending the cord, and disappeared altogether at the commencement of the first pair of dorsal nerves; while on the right side they remained nearly similar to those represented on the left side in fig. 28. The same state of degeneration affected the parts surrounding the central canal (*b″* fig. 28), and extended on the right side across the anterior commissure around an enlarged and unhealthy looking blood-vessel entering through the anterior median fissure (*s*).

(10) This condition of the grey substance, with that of the white columns last described, continued upward through the whole of the space from which the first dorsal nerves take their origin; the morbid area around the canal being larger for a few lines, as represented in fig. 29 (*b″*). From this a narrow streak of the same substance (*b′*) was seen to extend backward along the inner side of the base of the horn, on the left side. At the upper roots of the same nerve (first dorsal) this large morbid space almost entirely disappeared, the streak on the left side continuing, with some small patches on the right. Its decrease, however, was only for a few lines; for at the commencement of the eighth cervical nerves, it began again to extend itself, eroding, as it were, and destroying the under surface of the posterior white columns, which were softened to a considerable degree. Some minute spots of degeneration were seen here and there in other parts of the grey substance. These various lesions continued to a greater or less extent through the rest of this region (the eighth cervical), the morbid spaces along the inner edges of the posterior horns increasing and diminishing alternately on the opposite sides, varying in shape in almost every section, and encroaching,

generally, on the under part of the posterior columns, which were, therefore, in these instances, entirely separated from the grey substance. At the level of the seventh pair of cervical nerves, the degeneration diminished in extent, affecting chiefly the under part of the softened posterior columns, which were thus separated from the transverse commissure and inner sides of the bases of the posterior horns, by a transparent, narrow, and irregular streak.

Nearly the same condition of the cord was found through the rest of the cervical enlargement, only small streaks or patches of degeneration occurring here and there in the posterior cornua. On approaching the medulla oblongata, the degree of softening in the posterior columns diminished from within outward. Opposite the third cervical nerves, although all parts of these columns were softer than natural, yet the only portion that was actually pulpy, was that between the bases of the cornua, in the form of a longitudinal cylinder, which, on making a transverse section, completely separated from the rest of the substance, leaving the posterior commissure and inner bases of the posterior horns entirely uncovered. The grey substance was not nearly so much affected as in the lower part of this region; but the posterior commissure contained one or two small patches of degeneration, with a few corpora amylacea; while the anterior commissure was in some sections nearly destroyed by the same kind of lesion, and contained a few of the peculiar cell-like bodies constituting the oval tumour which was found in the posterior columns of the conus medullaris, and represented in fig. 3, Plate II.

(11) But besides the variety of morbid appearances which I have now described in different parts of the cord, one constant condition of the nerve-cells was observed throughout its entire length. Nearly all these bodies, in every section, were reduced to about one-half or two-thirds of their normal size, and were considerably altered in shape. They had also, to a variable degree, undergone certain changes in structure. The first indication of these changes was an unusual accumulation, around the nucleus, of yellow or brown pigment granules which either partially obscured or replaced it. Fig. 31, A, is an exact representation of a cell in this stage of transformation, taken with the others of the same figure, from a little below the middle of the lumbar enlargement. Here we find the nucleus enveloped and almost entirely obscured by a large ovoid mass of pigment granules, around which the remaining contents (*a*) of the cells have retained their transparent aspect and natural structure. The next step in the process of degeneration is the gradual

disappearance or wasting of these superficial or transparent contents (*a*) of the cell, and the consequent shrinking of the investing membrane or cell-wall, until it closely surrounds the central mass of pigment, as seen in the three cells represented at B. In consequence of this atrophy of the superficial parts of the cell, its processes become gradually thinner, and at length nearly obliterated. This was more or less the case with all the cells of the group, of which the greater number were in the condition represented at B. At C, the accumulation of pigment is at one end, and gradually fades away toward the centre, leaving the rest of the cavity transparent. When the sections were tinted with solution of carmine, only the transparent portions of the cells received the colour, the pigment masses, and consequently the whole of those cells which consisted entirely of them, retaining their yellow or brown hue. This alteration in form and structure was not limited to the nerve-cells of the anterior cornua, but was found also in those of the posterior vesicular columns, and of other parts of the grey substance.

(12) Through the lower part of the medulla oblongata, the posterior columns, although nowhere pulpy, were everywhere softer than in health, and contained several large fissures, in which irregularly-dilated blood-vessels were lodged. The grey sub-substance, immediately behind the canal, and which in this region is more than a mere commissure, contained several enlarged blood-vessels, and broke up readily under the most careful section. Through the rest of the medulla oblongata, all the central parts immediately surrounding the canal were involved in the softening process, but to a greater extent in front, and in some sections as far forward as the back of the anterior pyramids.

The olivary bodies were smaller than natural and altered to a certain extent in their elementary structure. Their lower two-thirds formed no projection whatever above the level of the surface. Internally, the grey laminæ presented their usually convoluted form; but the fibrous substance which they enclose, as well as their cells, was atrophied to a variable degree. In some of the convolutions the cells had almost entirely disappeared. In many sections, the transverse decussating commissure was involved in the softening process, and in others, the blood-vessel which enters between the anterior pyramids, as well as the beautiful series of parallel branches which it gives off to the bundles of this commissure, were here and there either enlarged or atrophied, and appeared sometimes as if artificially injected.

In the pons varolii, all the central parts were somewhat softer and more vascular than in health, and had very much the same general appearances as the corresponding parts of the medulla oblongata.

The cerebellum and its peduncles were of average size, and presented no deviation from their normal aspect and structure.

The fibres of the anterior nerve-roots, within the cord were evidently reduced below their usual diameter, in consequence of atrophy of the axis-cylinders; but outside the cord, the nerve-roots were apparently of their average size. This difference was probably due to the quantity of neurilemma surrounding the fibres external to the cord, as well as to their larger share of white substance, which perhaps is not affected by atrophy so readily as the axis-cylinders.

(13) It may be well to state, that all these morbid and abnormal conditions of the nervous centres were not only ascertained, but described and sketched before I knew anything of the history of the case, beyond the general fact that it was a case of muscular atrophy, accompanied by paralysis.

Now when we come to compare together the two series of facts it is gratifying to find that the symptoms which were manifested during life, are in almost every instance explained in a remarkable manner by the lesions of structure that were found after death. From the history of the symptoms we learn that both the lower extremities, and subsequently the abdominal muscles, became completely affected by motor paralysis. On examination after death we find that the whole of that region of the cord from which these parts derive their nerves was injured by disease. Thus to sum up, in a general way, the prominent lesions: in the conus medullaris nearly the whole of the grey substance and part of the posterior columns were replaced by a morbid and cylindrical mass. Through the lower portion and middle of the lumbar enlargement only part of the posterior columns were replaced by the morbid cylinder, the rest being unnaturally soft. A large proportion of the posterior roots were also involved in the softening, as was also the posterior part of the lateral column on the right side. The cells of anterior grey substance, and of posterior vesicular columns were more or less atrophied, and wholly or partially filled with pigment granules. In the upper third of the lumbar enlargement there was a variable degree of softening of all the white columns, but particularly of the posterior on right side, with softening and injury of the posterior commissure, from which they were separated. The posterior vesicular column on the

right side contained tracts or patches of granular degeneration; that on the left entirely disappearing in some sections. The form and symmetry of the grey substance was strangely altered in some sections. The anterior cornua were somewhat reduced in size; and their cells, more or less, were loaded with pigment granules.

We are informed that the paralysis of the lower extremities was followed by almost total loss of power in the arms. On examining the brachial enlargement there were found several dilated, and more or less degenerate, blood-vessels on each side of the canal, surrounded by transparent patches of granular degeneration, which involved the posterior commissure, and in some places the anterior. In certain parts of the brachial enlargement the patches of degeneration extended along the whole inner side of each cervix cornu. The posterior columns were more or less softened, particularly their deeper portions which rested on the posterior commissure. The cells of the anterior cornua were somewhat atrophied and loaded with pigment granules.

After paralysis of the lower and upper extremities came that of the muscles of the trunk. The first effect of this extension of the disease, we are told, was inability to sit in the erect posture, and then inability to strain at stool, or while passing water, and loss of power over the expiratory muscles, which rendered coughing impossible. In accordance with these symptoms we found that in the upper part of the lumbar region, whence the musculo-cutaneous nerve, which supplies the expiratory muscles of the abdomen, takes its origin, the grey substance was curiously altered in form and structure, while all the white columns, but particularly the posterior, were in a softened condition. Moreover, through the lower two-thirds of the dorsal region of the cord, which supplies nerves for some of the abdominal muscles, for the intercostal muscles and those of the back concerned in maintaining the erect posture, there were changes in the form and structure of the grey substance, enlargement of the blood-vessels, and softening of the posterior columns; while in the upper third of the same region several clots of extravasated blood were found in the substance of the anterior cornu, and extending into the corresponding tractus intermedio-lateralis. The grey substance was also curiously altered in shape and symmetry; the anterior cornu on the left side was smaller than on the right; the blood-vessels were frequently, but irregularly dilated; and the posterior white columns were unnaturally soft.

The last of the parts that became paralysed were the diaphragm and the respiratory muscles of the neck; and the patient died of very gradual cessation of the respiratory powers. On examination it was found that those portions of the cord which give origin to the phrenic nerves, the lower roots of the spinal-accessory, and the cervical plexus, were more or less injured by disease. They were less affected, however, than the parts immediately below. Now this is precisely what might have been expected from the previous paralysis of all the other respiratory muscles; for since the respiratory movements were now entirely dependent on these nerves, which, however, were barely competent alone to maintain them with sufficient activity to support life, death would necessarily ensue before any great amount of destruction could invade the grey substance from which they were derived.

But although, as we have seen, there is a striking general correspondence between the symptoms of the case and the lesions of structure in the nervous centres, there is one fact that does not appear to be easily reconcileable with the morbid anatomy. We read in the history that "sensibility was everywhere perfect." Now I have shown that in the middle of the lumbar enlargement the superficial halves of the posterior white columns, between and immediately behind the heads of the cornua, were quite pulpy, and that in this softened mass a large proportion of the posterior roots of the nerves were, of course, included. How can we account for the apparent discrepancy between these facts? We can only have recourse to one or the other of the following suppositions: 1st. That there must have been some failing sensibility in the lower extremities that remained unobserved. 2ndly. That the nerve-roots which traversed the softened columns did not participate in the injury. 3rdly. That the roots which enter through the posterior lateral fissure, and not through the white columns, are the only fibres engaged in transmitting impressions that excite sensation. In whatever way the difficulty may be explained, the retention, generally, of unimpaired sensibility, with general softening of the posterior white columns, goes to support the physiological conclusion, that these columns, if at all, cannot be the only channels for the transmission of sensitive impressions. This conclusion I arrived at in 1853 (Phil. Trans.) on anatomical grounds—from my own investigations into the course taken by the posterior roots of the nerves within the substance of the cord. Two years later the same conclusion was drawn by Dr. Brown-Séquard from his experiments on animals (Gazette Medicale and Comptes Rendus, 1855).

(14) With regard to the cause of the muscular atrophy in this case, it must of course be referred to the lesions of the spinal cord, for its appearance was subsequent to the paralysis. Moreover, there is good reason to believe that the wasting of the muscular tissue was not a remote or indirect, but an immediate or direct effect of those lesions, since it followed the paralysis too rapidly to be accounted for by mere inactivity of the paralysed muscles. In certain cases mechanical injury, exhaustive exercise of the muscular tissue, or an attack of fever, may be the exciting cause of the atrophy; and these facts, together with the supposed absence of disease of the spinal cord, have led to the general belief that muscular atrophy is not dependent upon lesions of the nervous centres. But even in such cases it is probable that the failure of nutritive reparation is due not to injury or exhaustion of the muscular tissue alone, but to injury of its nerves, or exhaustion of their supply of nerve-force, and the consequent disease or atrophy of that part of the spinal cord from which they are derived; or in cases occurring after fever, it may be due to injuries which the nervous centres have sustained during that disease. It is true that there is no apparent reason why atrophy of muscle should not be as independent a disease, as atrophy of nerve-tissue; but then it is a significant fact, that in every case of this malady in which the spinal cord has been properly examined, this centre has exhibited the most unequivocal evidence of disease. The first two were examined by myself according to the method which I then recommended. In one of them* there was no decided or complete motor paralysis, but only muscular weakness, which preceded the wasting. In those regions of the spinal cord which supplied the wasted muscles, I found in the grey substance, and generally around or in the vicinity of blood-vessels, numerous patches of transparent granular-degeneration of precisely the same nature as those which were found in the present case. Moreover, the nerve-cells contained an unusual number of coarse pigment granules, and corpora amylacea were abundant around the canal.

In the second case† the lateral halves of the grey substance were frequently unsymmetrical or mis-shapen, and contained numerous patches of granular degeneration; while the nerve-cells of the anterior cornua were nearly all atrophied or shrunk ot one-fourth or one-fifth of their original size.

The third case was treated and examined by Dr. Gull, and

* Beale's Archives, No. 9, vol. iii.

† British and Foreign Med.-Chirurg. Review, July, 1862.

described in Guy's Hospital Reports, vol. viii. 1862. There was progressive paralysis and flexion of several fingers of both hands, with subsequent wasting of their muscles. At this stage of the disease the patient sickened with typhus fever and died. On examination after death there was found in the cervical enlargement of the spinal cord considerable distention of the central canal, from accumulation of fluid within it, with consequent atrophy of the grey substance. In certain parts of this region there appears to have been absorption of nearly the whole of the posterior portions of the grey substance, only a certain extent of the anterior cornua remaining.

The fourth case is that which forms the subject of my present communication.

It is worthy of note that in the second case the symptoms began soon after a sun-stroke received in Australia. On examining the brain (which before I received it was reported as healthy) I found one portion of the marginal convolution of the longitudinal fissure to be much softer than the rest, and closely adherent to the membranes. In the present case also it has been seen that the symptoms made their appearance soon after a severe fall and blow on the back of the head, which rendered the patient unconscious for some time. It is not improbable that in both cases the shock to the cerebral centre may have been the exciting cause of the morbid changes which were found in the medulla oblongata and spinal cord. However this may be, it would be well in all future cases to note any accidental shock which the brain may have received, and examine this organ carefully after death. In cases also, occurring after fever, it might be important to inquire whether the attack was accompanied with severe cerebral, or other nervous symptoms. Indeed, in taking clinical notes, and making out the history of the disease, we should be careful to observe every circumstance that can possibly affect the case; for it is only by this close and minute investigation during life, with an equally searching and complete examination after death, that we can hope to arrive at definite and exact conclusions in both a pathological and physiological point of view.

THE FUNGUS FOOT OF INDIA.

(Abstract.)

In the "Intellectual Observer" for November, 1862, is a most important paper by the Rev. M. J. Berkeley, upon the above subject, to which we would direct the attention of the Profession.

We are daily enlarging the domain of our knowledge of the immense influence which organisms of animal and vegetable origin exercise in the production or aggravation of disease in the animal as well as the vegetable kingdom.

It is in our endeavours to trace these influences and connections that the microscope has rendered such good services to medical science.

The following is but an abstract of Mr. Berkeley's paper: for the further details we would refer the reader to the original.

It is well to bear in mind that the observations come to us with all the authority which the name of the ablest and most successful investigator in cryptogamic botany in this country can give them.

In March, 1860, Dr. H. V. Carter, Professor of Anatomy and Physiology, at Grant Medical College of Bombay, furnished his first report upon a disease, known in many parts of India as "Fungus Foot," or, scientifically, as "Podelcoma or Mycetoma."

The disease, which has hitherto occurred amongst natives only, is undoubtedly due to the presence of a fungus, which, by its growth, destroys the bones of the lower extremities, and induces death from exhaustion unless this be prevented by timely amputation.

The fungous matter assumes various forms, and Mr. Berkeley begins with the most typical.

1. The first case, then, is that in which the bones of the foot and the base of the leg bones just above the ankle, for the disease never ascends higher, are perforated in every direction with roundish cavities, varying in size from that of a pea to that of a nut or pistol bullet, the cavities being filled up with a dense fungous mass of a sienna red within, but externally black and resembling a small dark truffle. From these cavities, canals lead to the surface, from which a purulent fœtid discharge is poured out, often accompanied by little pieces of the fungus. The foot presents externally the peculiar turgid appearance which it so often assumes in bad cases of scrofula. Besides the canals, pink stains or streaks are observable on the

skin, and penetrating the subjacent tissues, filled with spherical or ovate groups of minute bright orange-coloured particles, and containing occasionally a few larger cells, the nature of which has not at present been ascertained, though it is conjectured that they present the earliest appearance assumed by new attacks of the disease.

Of the structure of the large truffle-like bodies Mr. Berkeley is enabled to give an excellent figure made by Dr. H. J. Carter, from a case which he examined immediately after amputation. The parts in which the structure is most visible present precisely the characters of a true Oïdium, such as O. fulvum. Short beaded tawny threads arise from a common base, consisting of cylindrical articulated filaments, having at their tips large spore-like cells. These, however, do not appear to germinate in situ, but to become enormously dilated, their albuminous contents assuming at length a resinous consistence, while many of them burst, and nothing remains except fragments of the old cell-walls. The resinous matter is inflammable, but its exact chemical nature has not yet been ascertained.

If, however, these large terminal cells be a form of fruit, they are not in all probability the only form, for on three occasions the black fungous masses, either in maceration in water, preserved in alcohol, or scattered purposely on rice paste, to see if any further development would take place, gave rise to a peculiar mould, which it can scarcely be doubted is the perfect condition of the species, though at present it has not been observed in any other situation; a circumstance, however, which need excite no surprise, as so little attention has hitherto been paid to the minute fungi of India. Of this fungus a sketch from Dr. H. J. Carter's original drawing exhibits its beauty and singularity. The change from the early yellow or colourless transparent threads and sporangia to a fine red or crimson, make it a most lovely object for the microscope.

The fungus made its appearance only in the months of April and May. Mr. Berkeley says that he has great hopes next spring to be able to cultivate the species from specimens placed in his hands, as so many moulds are grown with the utmost facility on rice paste, which affords an admirable opportunity of examining them in every stage of growth, proper caution being taken not to confound different species with each other, as several will sometimes appear together, or in succession on the same mass of paste.

The fungus resembles closely the genus Mucor, but there is no columella in the sporangium—a character which accords with Chionyphe rather than with Mucor. Indeed he does not

see a single character in which it differs generically from Chionyphe, though the two recorded species occur only under snow. It is very possible, however, that the proper habitat of our fungus may be upon damp soil.

Mr. Berkeley then gives an admirable and minute description of the fungus and its mode of germination.

The species, he says, may be characterized as Chionyphe Carteri; hyphasmate ex albo flavo rubroque; sporangiis denum coccineis; sporis breviter fusiformibus.

Mr. Berkeley, in speaking of the pink streaks mentioned above, says:—It is highly probable that many of our common moulds occasionally commence with a similar condition. The first indications of vegetation on tainted meat or paste assumes the form of little gelatinous spots of various colours, consisting of extremely minute distinct cells, and these seem to be an early stage of common species of Aspergillus and Penicillium, or other genera. If there be any truth in the notion, which he has entertained for some time, that hospital gangrene depends upon some vegetation of this nature acting as a putrefactive ferment, there may be good reason for believing that the red spots in question are really the commencement of the disease under consideration.

2. We now come to a second form under which the disease appears. In this the black fungous masses are entirely wanting, and in their stead masses are found of what looks like sloughing tissue. White granules, however, occur in the cavities and in the discharge, which appear to be a form of the same fungus, though the identity has not been proved. Under the microscope it wears the appearance of a congeries of large cells filled with smaller, much after the manner of Microhaloa firma, as figured by Kützing. Whether the perfect form of the plant be the same or not, the phases of the disease produced by it are exactly the same, and the malady admits of no other remedy.

3. A third case is known under the name of the Madura foot, from its having occurred at Madura. In this case the foot becomes enormously enlarged about the instep, though not so much at the ankle, while the toes are hypertrophied, and almost lost or imbedded in the mass. The small bones are nearly destroyed, leaving behind a pallid or reddish tissue, while the others are more or less excavated. There are the same canals and external sanious apertures. In some parts they are filled with the same fleshy tissue, in others lined with it, where large cavities are formed by the junction of several canals containing broken up osseous tissue from the exposed bones around, grey

fragments and masses of pigment. The pink colour is partly owing to a general diffusion of pigment which tinges the oil-globules, and partly to the presence of very numerous single or aggregated elliptic particles. These granules are from the fiftieth to the 1-130th of an inch in diameter, and occur sometimes as single ellipses, sometimes as two combined at the extremities of their major axes, and sometimes as square bodies with rounded extremities divided crucially into four. They do not seem to be cells, at least cells of cellulose, containing a grumous mass, but resemble rather certain Palmellæ. They are quite visible to the naked eye, insomuch that when the sawn surface is first exposed to view, it appears as if strewed with grains of red pepper. They are similar in essence to the granules described in the second form. None of the black fungous masses appeared, but they were globular opaque bodies of various sizes, which, though at first apparently so different, are closely connected with the fungus of the first form.

The foundation of these bodies consists of one or more large mother-cells filled with a mass of daughter-cells. The structure often so exactly simulates that of minute moulds, that it is very difficult to get rid of the notion that they are really vegetable growths. Pure sulphuric ether, however, dissolves them completely, and shows that they are merely different forms assumed by stearine. The foundation is, however, in every case an organised cell, the red colour of whose daughter-cells is precisely that of the oidioid thread of the black fungus.

There is not the slightest ground for supposing—says Mr. Berkeley—that the disease depends on inoculation with the spores of any of the truly parasitic fungi belonging to the tribe of rusts and mildews, and, therefore, more or less closely allied to Æcidium; but great reason, on the contrary, as appears from what has been stated above, for looking to the origin amongst the mucors, even were there not something like direct proof.

It is well known that mucedinous fungi make their appearance within cavities in vegetables which have no apparent connection with the outward air. Nothing, for example, is more common than to find a pink mould (Tricothecium roseum) in the middle of a nut; and an allied vegetable production (Dactylium oögenum) has been found in an unbroken egg. Even the cells of plants themselves produce fungi which fructify within them. How the spores are carried there is at present a mystery, which may some day be cleared up, like the origin of many intestinal worms, which can no longer be brought forward as an argument for equivocal generation. There is, however, reason to believe that amœboid

growths are not confined to such dust-like fungi as the Æthalium which is such a pest in pine-stoves; and zoospores have already been ascertained to occur in certain moulds, as, for example, in the Peronospora which causes the potato murrain. The Fungus foot is confined to the natives who go about with naked feet, and the spores might easily be introduced through some scratch, even were it impossible for them to penetrate by the pores of the skin. When once introduced beneath the cuticle a single spore might soon perform the work of destruction spreading in every direction, and according to the peculiar condition of the secretions, the mycelium might put on a hundred different modes of growth. Besides, if the fungus is capable of causing the absorption of solid structures like bone, it is easy to conceive that a spore in contact for some time with a moist foot might penetrate the cuticle simply by absorption. Cleanliness in the first instance seems to be a preventive, but when the fungus is once established, there seems to be no cure save amputation—which, happily, when resorted to in time appears to be completely successful, as the disease never spreads beyond a certain point, though, if it be allowed to take its course, death will ensue from the exhaustion consequent on pain and the continuous discharge.

GONORRHŒAL RHEUMATISM.

By Jeffery A. Marston, M.D., Royal Artillery.

Rheumatism is a distinct and very peculiar complication of gonorrhœa. It attracted the attention of Swediaur, and has been described by various observers.

It very rarely indeed appears to attack the female.

Sometimes it happens, that rheumatic symptoms follow after every gonorrhœal attack, as in the case related by Sir Astley Cooper.

Some pathologists perceive in these rheumatoid affections strong proof of a specific virus as the cause alike of the local urethral disease and, by its absorption, of any articular or ophthalmic symptoms that may appear. If by specific be meant a syphilitic virus, the writer conceives that there is an absence of any proof of this in the bare fact of the occurrence of symptoms so entirely dissimilar to those constitutional ones resulting from syphilitic inoculation. As

the phenomena of rheumatism have been also known to follow urethritis from various and dissimilar causes, it is obviously not due to any one specific inflammation of that part.*

The causes are very obscure. By some it is held, that the explanation is to be found in the supposition of a gonorrhœa attacking one of rheumatic diathesis; but this does not seem to be at all proved.

Dr. Wilks conceives the rheumatic disease to be due to a subacute form of pyœmic inflammation, and that it owns its origin to a very similar, if not allied cause. In this view the writer is inclined to concur, but it has its difficulties, viz. :—The disease is rarely, if ever, dangerous, or connected with other symptoms of pyœmia; it is also of uncommon occurrence, save in those persons whose proclivity to take rheumatism after every gonorrhœa amounts almost to an idiosyncrasy. There are no good grounds for supposing the affection of a joint to be due to a metastasis of inflammation from the urethra.†

Gonorrhœal, like ordinary rheumatism, appears under two forms, according as it attacks the fibrous tissues and muscles, or the synovial membranes of joints.

The first occurs as a subacute inflammation and painful affection of the fibrous tissues of some part of a limb, particularly the sheaths of the thicker groups of muscles, such as those of the hip, front of thigh, shoulder, &c. An affection of the sclerotic and fibrous coats of the eye—the conjunctiva participating in the morbid action—may be included in this form.

The second comprehends the arthritic

Of the various joints attacked, the knee is the most frequently so; the ankle and wrist joints are also very liable to be affected. Other joints may be attacked, and the disease need not be limited to one, although it often is.

These two forms may be combined.

The writer can corroborate the experience of Ricord, that a painful affection of the plantar region is not uncommon; and here it appears, that not only the fibrous fascia, but the ligaments and tissues entering into the various tarsal joints may be affected.

* Since this paper was penned the writer has perused the very practical lectures by Dr. Fuller (Lancet, January 31st, 1863).

Dr. Fuller considers that the disease has nothing in common with ordinary rheumatism, except pain in the limbs and occasional swelling of the joints. He thinks that it is the result of a specific virus obtained from some peculiar form of vaginal poison distinct from that which occasions ordinary gonorrhœa.

† Among the hundreds of cases of gonorrhœa that army medical practice supplies, it is remarkable how rarely rheumatism seems to follow.

The disease differs from ordinary rheumatism in wanting the inflammatory fever, in the absence of a marked tendency to sweating, in the infrequency of an extension to various joints, and in the freedom of the patient from the occurrence of peri, or endo-cardial diseases.

It appears, as a rule, after the inflammatory stage of gonorrhœa has subsided; sometimes it may be regarded as a sequela of the urethral affection, inasmuch as it does not arise until after apparent recovery, or during the blenorrhœal stage of the disease.

Symptoms. Premonitory symptoms may be very slight or entirely absent, and it is rare indeed that the implication of a joint is ushered in by more than a trifling pyrexia. After vague muscular or arthritic pains it may be, the patient notices a stiffness and swelling of one joint—the knee for instance. Upon examination this is found swollen, with effusion into synovial membrane. The local temperature is slightly increased, but the skin is not altered in colour, although it sometimes happens, that there is an erythematous blush upon it. As the disease becomes more advanced and chronic, the areolar tissue may swell with slight œdema, and the structures surrounding the joint may become thickened.

When the ankle joint is attacked, the swelling is apt to appear unilateral; that about the outer malleolus being commonly the most marked.

The amount of pain varies considerably in different cases. It is greatest in the fibrous or muscular form, and in both is prone to nocturnal exacerbations. When the joints of the, tarsus and plantar fascia are involved, pain is considerable, particularly on movement; when the knee-joint is implicated the pain may be inconsiderable.

The ocular affection—distinct from the purulent conjunctivitis, ordinarily termed gonorrhœal ophthalmia—is generally attended with much pain.

These affections are eminently chronic, prone to relapses, and but little amenable to treatment. Fortunately, however, they generally terminate by resolution and absorption of any effused fluid; the joint remaining often weak and stiff for some time afterwards. Suppuration, ulceration of cartilage and bone are rare indeed from this cause alone; but various morbid processes may be excited in the debilitated or strumous by an attack of gonorrhœal arthritis. Chronic thickening of the synovial membrane and ligaments may be produced, leading to anchylosis or impaired and painful mobility of the joints.

Treatment. The first object to be attained is—the cure of

the urethral discharge,—by means of injections, and the occasional use of a bougie.

As to the remedies found so useful in rheumatic affections, these may at once be dismissed as useless; and the same may be said of anti-blenorrhagic agents.

It is a good plan, in all cases, to endeavour to amend the general health by attention to diet, and such measures as the different states of the patient will indicate. The muriated tincture of iron, given after meals, is occasionally very useful with this end in view.

The iodide or bichloride of mercury, in tincture of bark, appears to be of real avail; iodide of potassium ought to be given at the same time, provided that it does not keep up the urethral discharge,—which it has a tendency to do.

If the pain be great, which is very likely to be the case when the eye is affected, a full dose of Dover's powder or pil. saponis c. opio, at bedtime, had better be given.

Among the local measures which the writer has found, by far, the most useful are,—the application of douches of cold or tepid water to the affected parts, afterwards rubbing them dry, applying a strong solution of iodine, and wrapping them in cotton, wool, or flannel.

If the parts affected be tender, or the local temperature decidedly increased, the writer applies fomentations, until these symptoms are subdued.

When one joint only is affected, or when the seat of pain is fixed and well defined, blistering, of some kind, affords relief.

There is no necessity to affect the patient's system by the internal use of mercury, as above recommended, nor by the external application of the mineral, but the writer commonly combines mercurial ointment with ol. crotonis, and directs the patient to rub the integument with this, as a means of obtaining the benefits of counter-irritation.

When the disease becomes chronic, and thickening and stiffness only remain, if cold douches and frictions produce no change, the best course is to compress the joint by means of strips of adhesive plaister, a sufficient number of layers being applied to maintain equable and firm pressure.

ON CASTS COMPOSED OF MUCUS FORMED IN THE STRAIGHT PORTION OF THE URINIFEROUS TUBES.

By Lionel S. Beale, M.B., F.R.S.

Plate VI., Fig. 31.

There are certain casts which are not uncommonly found in the urine in cases of renal irritation, which differ in important particulars from the casts usually present in renal diseases, and have not been described. They consist of a slightly granular viscid matter which often becomes completely disintegrated within twenty-four hours after the urine has been passed. They seem to be composed of mucus, and when there are not many, they may easily be passed over as they are extremely transparent. But if it so happens that the urine is rich in urates, the precipitate is deposited in the mucus, and thus the cast becomes a very distinct object. Albumen does not exist in urine which contains mucus casts, and I have examined specimens of urine in which such casts were very numerous indeed, without detecting a trace.

I have found them often enough in the urine of persons suffering from renal calculus, and in several cases of what might be termed renal irritation, without there being any distinct evidence of the existence of calculus. A specimen of urine sent to me by Mr. Hawkins contained a bulky semi-transparent deposit, consisting entirely of delicate shreds. Upon removing some of these from the bottle, and floating them out in a little water, over a piece of dark paper, they were seen to consist of numerous delicate shreds radiating from a point in a V like form. They were clearly moulds of the straight portions of the uriniferous tubes. A small portion of one of these masses is represented in Plate VI., Fig. 31. Sometimes when a kidney is injected with strong gelatin, shreds exactly resembling these will escape from the tubes.

The importance of not mistaking these mucus casts for the transparent casts of the uriniferous tubes formed in chronic disease of the kidney, need scarcely be dwelt upon. No special treatment is required, beyond rest for a few days and diluents. Such casts are not often met with in the urine for any length of time.

THE NORMAL POSITION OF THE EPIGLOTTIS AS DETERMINED BY THE LARYNGOSCOPE.

By George D. Gibb, M.D., M.A., Member Royal College of Physicians London; Physician to the West London Hospital.

The mechanism of the various parts which enter into the construction of the larynx, and the physiology of the human voice, have long engaged the attention and study of physiologists and men of the highest philosophical talent. The labours of Professor Willis, Sir John Herschel, Bishop, and others in this country, together with many men of great scientific attainments on the continent, have shown the intricacy and difficulty attending the solution of the various questions connected with phonation, and the right interpretation of the production of the various sounds through the larynx. As the delicate organs which give rise to the latter, could not heretofore be seen in action in the living being, in a natural state, many of the hypotheses brought forward to explain some of the phenomena associated with phonation, were necessarily conjectural, and, therefore, enshrouded in doubt, because of the absence of positive proof. What has hitherto remained in doubt and uncertainty, now stands a fair chance of being satisfactorily worked out and rescued from obscurity by the application of an instrument, first invented in this country, for the actual inspection of parts that have hitherto remained concealed from the eyes of man. The publication of the "Researches on the Human Voice with the Laryngoscope," by Professor Garcia, in the Proceedings of the Royal Society, and the work of my distinguished friend, Professor Czermak, of Prague, "On the use of the Laryngoscope in Physiology and in Medicine," and its translation into the English and French languages, have given an impulse to the study of these various parts, which could scarcely have been anticipated. The desire to see and to learn the movements of the larynx and its connexions, in a state of health and disease, has been seized upon with avidity by all those whose attention was heretofore directed towards their study. The phenomena attending vocalisation, as witnessed in the action and vibration of the vocal cords, in the utterance of notes and sounds, in breathing, and other conditions, have, perhaps, engrossed the largest share of attention. Whilst not unmindful myself of these attractions, I have devoted some attention to the inspection of the parts above the glottis, more for the purpose of explaining some of

the anomalous and peculiar sensations so often experienced by persons labouring under abnormal conditions, than for any other reason. To carry out my investigations with success, I have examined as many healthy individuals as opportunities were afforded me of doing, with the view of ascertaining what was really the natural condition of things as seen in the living, above the glottis.

When these observations were first committed to paper, and brought before the British Association for the Advancement of Science, at Cambridge, on the 4th October, 1862, I had inspected the glottis of 300 healthy persons, but since then the number has increased to 530, and I am now prepared to submit my results, very briefly, for the consideration of scientific men. They are mostly confined to the condition and position of the epiglottis, and seem to be so important, that they have led me to form certain conclusions, which shall be presently noticed.

The influence of musical and other sounds upon the larynx, which has been studied at the same time, formed the subject of a lecture which I delivered before the Musical Society of London on the 11th of March.

But I must first be permitted to ask, what has been the received and acknowledged opinion heretofore in regard to the normal position of the epiglottis?

It has always been assumed that, owing to its peculiar formation, and the structures entering into its composition, the epiglottis invariably maintains the vertical or erect posture. This has never been disputed, and comes to us, I may truly say, uncontradicted from the earliest anatomists, from the time of Aristotle downwards. To refer, however, briefly, to a few of the more modern opinions:—

Professor Harrison, of Dublin, in his admirable work on anatomy, describes the epiglottis as very flexible, and extremely elastic, as it is easily depressed in deglutition, and its elevation is rapid, not requiring any muscular action, when once the tongue has advanced, and the larynx descended.

That able anatomist, Dr. Knox, whose loss science has had so recently cause to mourn, says that, "In general the position of the epiglottis is vertical, but it changes and becomes horizontal during deglutition; it serves, therefore, to cover and protect the superior aperture of the larynx during the deglutition of a solid and liquid aliment." *

In the article on the Normal Anatomy of the Larynx, in the "Cyclopædia of Anatomy and Physiology" (vol. iii, p. 103),

* Manual of Human Anatomy, 1853.

by Mr. Bishop, he observes, "the direction of the epiglottis is vertical, except during the act of deglutition, when it becomes horizontal."

In describing the act of deglutition, Professor Dunglison, who is the personification of the opinions of all who have preceded him, says that as soon as the bolus has cleared the glottis, the larynx descends, the epiglottis rises, and the glottis opens to give passage to air.

According to Meckel, its elasticity and ligaments cause it generally to lie perpendicular, and to rise towards the isthmus of the fauces.

Cloquet shows that the direction of the epiglottis is liable to vary in the different circumstances of life. It is vertical in the most common state, but remains horizontal when the food passes from the mouth into the œsophagus.

And Professor Czermak declares M. Meyer to be in error in asserting that the larynx is closed or is covered by the epiglottis depressed mechanically by the alimentary bolus, although he acknowledges that he is right in stating that after the passage of the bolus, the epiglottis is raised again by its own elasticity and that of its ligaments.

These opinions are quite sufficient to show what the prevailing view has been up to, I may say, the present hour, even with Professor Czermak, and it is but fair that I should ac knowledge that it applies to the largest portion of mankind.

In the course of my investigations of the physiological use of the laryngoscope, I discovered that there were individuals whose epiglottis was not elevated, but naturally depressed, and had been so from birth. In other words, that it was an abnormal physiological condition, so to speak, for there was really no evidence to prove the existence of any disease past or present giving rise to it, and the interior of the larynx could not be seen but with great difficulty, from the absence of the erect position of this fibro-cartilage. Very probably if this condition has been noticed by others in the healthy, it has been set down to relaxation of the proper ligaments of the epiglottis, or to some state allied to disease. In the large number of healthy persons examined, I satisfied myself that it was not so. Many of my inspections were sometimes made on single individuals, at others on a number in the presence of a company of persons taking all indiscriminately. In this way, I have examined parties of from six to ten and twenty persons on single occasions. They included several young children of both sexes, and persons as old as sixty, and a few upwards to the age of ninety-two. I have seen the larynx in children at the first examination, and it

is astonishing how well they comport themselves under the influence of example; and I may state that the vocal apparatus in the child is one of the most beautiful objects that can come under the notice of the physiologist.

In a certain proportion of the 520, that is to say, eleven per cent., had an absence of the erect position of the epiglottis, and I was enabled to determine in a few that it actually was congenital, for whilst the parent had this condition, so had the child, and yet in neither had any inconvenience that I could determine, been experienced.

Although I have frequently examined the epiglottis in the dead as well as the living, I was not aware of the circumstance, until I read it in Dr. Türck's book on the Laryngoscope (French edition), that in the dead the epiglottis projects backward more than in the living, and the plates published in his work illustrate this, for they were taken from the dead. Dr. Türck considers it necessary to remind the reader of his book that it is important for him to remember, that in the living individual, the epiglottis seems less strongly projected backwards, and that in consequence, the interior of the larynx can be seen more readily than in the dead; and this he had confirmed by numerous laryngoscopic observations made upon the dead.

This fact renders my own observations only the more important, for the depressed condition of the epiglottis occurring as a natural state, produces such alterations in phonation, and such inconveniences in the event of disease, together with rendering the individual so circumstanced predisposed to incur serious pathological changes, that an early examination of the larynx in the young, s h o u l d b e a m a t t e r o f n e c e s s i t y, as a guard against future evils, as much so indeed, as vaccination is a preventive or modifier of small-pox.

The physiological modification of phonation, the result of backward epiglottic projection, is an absence of clear intonation, so usually heard in young persons. Vocal utterance is thicker, deeper, and more guttural in character, and as far as I could make out, the persons thus placed were not singers, unless to a moderate degree. One lady who had her epiglottis placed in the manner described, and who was not aware of it, until I discovered and explained it to her, assured me she always enjoyed fair health, but that at times her throat felt tired. Her daughter, a girl of 16, with her epiglottis precisely in the same position, was delicate when young, but I could not ascertain whether she ever had any throat derangement.

The condition of the epiglottis in the 520 persons examined, varied much according to age and sex; when vertical it pos-

sessed the natural shape and colour in nearly all, being modified in regard to the former in the young of the male sex, and in females up to 30, and a few upwards. Whilst in the elderly, in some it was thinner, curled laterally forwards, and in colour somewhat altered to grey or drab. As an invariable rule, the interior of the larynx could be seen with greater facility in the elderly than in the young. In those again in whom the vertical position was absent, the cartilage was thinner, much thinner, flatter, and broader, and in some it appeared to excite a constant secretion of mucous between it and the sides of the glottis, with which it was in contact. As compared with the vertical, even in the young, it had not the plump and thick appearance which is so striking in the vertical. In one instance of nearly horizontal position in a lad of 18, who was the subject of fissure through the soft and hard palate, it possessed the natural appearance and thickness, and was itself slightly fissured.

The attenuation of the epiglottis in these cases is, I think, sufficiently explained by its exposed position, and arrest of action; it resembles a muscle that has become wasted from the want of use, or the absence of any power to excite it to contraction; and in the event of disease, we can imagine the further risk to which it is exposed, not to say that from injudicious topical medication.

We will suppose, for an instant, that a person is being treated for a throat malady, with the epiglottis laying almost flat upon the glottis, and that without making an inspection of the parts, a sponge probang is introduced with the intention of passing into the larynx. What would be the effect of such a proceeding upon the person undergoing the operation? The glottis would be almost completely closed by the pressure of the sponge upon what is here the upper surface of the epiglottis, and suffocation is for a short time imminent. Of several instances which have come under my immediate observation for diagnosis, this condition of the epiglottis obtained, and the sponge probang had been introduced several times before the persons came to me, whose glottis had never been inspected by the laryngeal mirror, and their sufferings during and after the operation were described as being severe in the extreme. Not only is their misery augmented in undergoing this infliction, but the upper or anterior surface of the mucous membrane covering the cartilage, and most likely the cartilage itself, is severely injured.

My present belief is, that if this position of the epiglottis has resulted from disease, there is a possibility of rectifying it,

but if it is a normal congenital peculiarity, then I am afraid it is almost irremediable, as the ligaments and muscles attached to it, are so arranged that it is next to an impossibility to alter them, until further light has been thrown upon the subject.

The question might be asked, was the epiglottis seen to project backwards or become depressed during examination, as the result of deep or grave vocal intonation? and might not this have been mistaken for the permanently depressed position? In reply to this I would give the following quotation from Mr. Bishop's paper lately published in the Proceedings of the Royal Society, "Observations made in the movements of the Larynx, when viewed by means of the Laryngoscope." (Vol. xii, No. 50, p. 144.)

"It is observed that while the pitch of the tones of the voice becomes more grave, the epiglottis is depressed, and the pharynx is relaxed; and, on the contrary, as the pitch becomes more acute, the epiglottis is raised, and the pharynx becomes contracted; the depression of the epiglottis probably assists in deepening the pitch of the vocal tube in the same manner as the lid of an organ pipe does."

I would remark upon this, that the depression of the epiglottis in the manner described by Mr. Bishop is comparatively slight, and by no means so complete during intonation as was seen in the cases I refer to; moreover, the epiglottis was seen depressed, without the utterance of any sound, and, therefore, could not depend upon any movement of the tongue, larynx, or pharynx.

In the computation of the percentage of cases wherein the epiglottis was naturally depressed, I have excluded a number which came under my observation for pathological investigation, and as the sufferings of those so unfortuately placed were discovered to be so remarkably great, I concluded, from the general symptoms and previous history, that in several, this condition of position was congenital, and hence the explanation of the misery to which they were subject. Then, again, in a proportion of those who were healthy, there seemed to be evidence of a disposition to derangement of the throat, and probably if the history of early youth could have been obtained in many it would have shown much trouble, risk, and danger experienced whilst passing through the diseases of childhood. I can imagine the effects of an almost flat epiglottis in such diseases as pertussis, rubeola, or scarlet fever. It seemed to me then, that the position of the epiglottis was a matter of the highest importance to the welfare of the young; its determina-

tion at an early period, as desirable as it is essential, for the prevention of future evils, and its neglect an evil of grave consequence. I have preferred bringing these facts, as briefly as the importance of the subject would admit of, before the profession, and if it has not been overrated, should be glad to obtain support in drawing attention to it with somewhat more of authority than could be expected from one individual—no matter how great his experience might be.

To sum up my conclusions, in regard to the epiglottis, it may be said :—

1. Physiologically speaking, the epiglottis is vertical in the great majority of mankind, in a certain proportion it is oblique or transverse.

2. The evils likely to arise from the latter at present appear to be so inconvenient, that it would be desirable that an inspection of the glottis should be made in every child where practicable, between the ages of four and ten years, for the purpose of ascertaining its correct position.

3. If it is found to be not vertical, a knowledge of the fact will prove beneficial through life in guarding against evils likely to arise, during the prevalence of epidemic sore-throat, or other diseases likely to involve the larynx.

4. No interference with the throat or larynx should ever be permitted without the aid of laryngoscopic inspection.

5. Whilst any imperfection in the voice or speech may be explained by the position of the epiglottis, independently of the vocal cords, a chance for the improvement of both is held out, by adopting some means that shall render this valve more oblique in direction than transverse, or possibly (but at present very doubtful) restore it to a vertical position.

It has not been my wish nor desire, to say anything in this communication on the subject of phonation originating in the action of the vocal cords, but simply to allude *en passant* to the modification of sound through the position of the epiglottis.

CASE OF ANEURISM OF AORTA, COMMUNICATING WITH THE PULMONARY ARTERY.

By Jeffery A. Marston, M.D., Royal Artillery.

Plate VII.

D—— M——, æt. 27, was landed at Portsmouth 21st January, 1862.

Previous History.—One day he helped to man a boat, he

pulled a very long distance as hard as he could, and, owing to the excitement, continued the exertion beyond "what he felt to be good for him." Upon landing, he became very faint, spat much blood, and had some brandy and water given him. He then went to hospital. His breathing became difficult, and he could not lie down in bed with any comfort. Dropsy afterwards appeared. Previous to the date of these occurrences he was perfectly healthy. Report sent with him states that he is suffering from "rheumatic carditis."

Present appearance and examination. General aspect; eyes bright, prominent; cardiac expression of face; lips and face dusky (but not markedly so). Anasarca and ascites. Veins upon the belly not prominent. Respiration costal (superior costal), 29. Slight venous regurgitation in jugulars. Carotids visibly pulsating. Radial pulse of water hammer character, about 115—120, &c., but not so frequent as the cardiac impulse. Chest (vide pen and ink sketch, outline figure, Plate vii.). Crepitant and loud wheezing râles over anterior and lateral aspects, (the back not examined, as the position required caused him so much discomfort). Cardiac dulness much increased. Apex beat at two seats—one an undulating impulse at ensiform cartilage; 2nd, a heaving impulse at seventh rib, below nipple. Upon the anterior aspect, over the base of heart, a purring tremor was appreciable to the hand. The heart's action was so quick and irregular as to defy an accurate analysis; this, however, is the best account of what could be made out:—

At the apex, beneath nipple, a loud soft systolic murmur; no second sound, but an indistinct murmur. At ensiform cartilage a faint systolic murmur. Over the transversely dull cardiac region a double murmur, harsher than at apex. At the base, the diastolic murmur was loud and marked, the systolic softer in character. This double bruit was audible upwards to the right sterno-clavicular articulation and there lost, as well as upwards to the left, as far as a little above the second rib.

No evidence of atrophy of the liver by percussion, rather the reverse. The lower and lateral aspects of abdomen dull; fluctuation appreciable (ascites). Epigastro-umbilical regions tympanitic. After having been treated for some weeks with slight, though temporary, relief, and twice tapped, he died, apparently of cardiac apnœa.

Post Mortem. Pericardial sac distended with fluid, but there were no traces of pericarditis.

Heart enlarged, with rounded apex from the dilated right venticle; weighed 20 ozs. Right side full of fibrin clots,

closely interlaced with the valves, columns and cords. Left side; dilatation of left auricle, its cavity containing some dark clotted blood. Mitral opening very large; nearly two inches in diameter; valves not diseased. Left ventricle dilated and much hypertrophied. One of the aortic valves was involved in an aneurismal dilatation of aorta, arising immediately after the exit of the vessel from the heart. This intrapericardial aneurism sprang from the antero left-lateral aspect of the aorta. The walls of the aorta were connected by adhesion with those of the pulmonary artery. Immediately above and to the left of anterior valve (which was adherent to the lining membrane of aorta) was a circular hole, the size of threepenny piece, communicating directly with the channel of the pulmonary artery. The edges of the aperture were indurated, and bevelled off towards the inside of the aorta. Lungs large, heavy, congested, as well as tougher than normal (cardiac lung). Liver nutmeggy and somewhat enlarged.

No other appearances, as bearing upon the nature of the case, require remark. From the history of this case it is pretty clear that an aneurism of the intra-pericardial portion of aorta existed, which dated its communication with the pulmonary artery at the period of the oppression, faintness and hœmoptysis following the exertion of rowing.

In a case wherein the heart's action is so excited an accurate analysis of the sounds cannot be attained with certainty. The diagnosis from this cause as well as from the fact that the murmurs generated at the aorta were conveyed downwards towards apex, could not be made. This combination of circumstances was not present in the only case upon record as diagnosed during life, viz., that related by Dr. Wade, of Birmingham, in the Medico-Chirurgical Trans., vol. xliv. From that paper I gather that this lesion is not so very rare. Of 18 cases of varicose aneurism recorded by Dr. Thurnam, 11 had perforated the pulmonary artery, and only 7, other parts, even including cases of communication with the superior and inferior cavæ.

Of 29 cases collected by Dr. Sibson,

17	opened into	pulmonary artery,
6	„	right auricle,
3	„	right venticle,
3	„	left venticle.
Total .. 29		

AN ABSTRACT OF THE CASES TREATED IN THE MEDICAL WARDS OF KING'S COLLEGE HOSPITAL FROM 1st JANUARY, 1863.

By Morris Tonge, M.B., M.R.C.P., Associate of King's College; Medical Registrar to the Hospital.

All the cases are reported. The most important cases are preceded by a *. The result and the treatment are printed in *Italics*. The cases have been arranged in groups.

Rheumatism—Acute.—J. A. æt. 16. Printer's reader. Adm. Dec. 25, 1862. Disch. Jan. 7. In H. 13 days. *Recovery.* Dr. Budd, vol. 74, p. 141. First attack. Prev. ill. 6 days (after getting wet through). Shivering, and pain, and swelling of right knee (8 days) and on 8th day right ankle inflamed (4 days). Heart unaffected. Highest pulse 104; highest resp. 24.

Alkalies quinine, opium, and blisters. Then sulphate of iron, sulphuric acid and quinine.

*Rheumatism—Acute.—T. W. æt. 28. Pressman. Adm. Dec. 2, 1862. Disch. Jan 17. In H. 46 days. *Recovery.* Dr. Budd, vol. 73, p. 105. Previous health good. Father subject to rheumatism. Very anæmic. Prev. ill. 14 days, with rigors and acid sweats. Commenced in left ankle, knee, hip; then joints of right leg in same order. Arms affected on 12th day. Pain in left side and pleuritic rub on 18th day. Systolic bruit over apex of heart.

Alkalies, quinine and opium; brandy 4 oz., and blisters. Fer Reduit. during convalescence.

Rheumatism.—S. D. æt. 7. Adm. Dec. 31, 1862. Disch. Jan. 17. In H. 17 days. *Recovery.* Dr. Beale, vol. 5, p. 92. Epileptic fits 1 month before. Prev. ill. 1 week. Shivering, profuse sweating, wrists and knees affected. No cardiac disease. P 112.

Alkalies and opium; and quinine, sulphuric acid, and sulphate of iron during convalescence.

*Rheumatism—Acute.—E. W., æt. 24. Laundress. Adm. Nov. 24, 1862. Disch. Jan. 2. In H., 39 days. Dr. Budd, vol. 73, p. 98. *Recovery.* First attack. Prev. ill. 14 days, with shivering, and pain in right knee. Remission of symptoms till 4 days before admission, when knees and ankles were attacked, and fever increased. Systolic bruit over apex on 22nd day. Highest pulse, 104.

Bicarbonate and nitrate of potass, and decoction of bark; wine, 3 oz., and blisters above the affected joints.

Rheumatism—Gonorrhœal.—G. B., æt. 42. Ballad Singer. Adm. Dec. 23, 1862. Disch. Jan. 28. In H. 36 days. Dr. Budd, vol. 73, p. 127. *Recovery.* Intemperate. No family history of gout. Prev. ill. 5 months. Inflam. of right great toe and ankle. Gonorrhœa. Pain and stiffness of neck and shoulders. Tympanitis and constipation. Oxalate of lime in urine.

Alkalies and opiates. Then with quinine and sulphuric acid, and assafœtida. Purgatives.

Rheumatism—Acute.—E. M., æt. 25. Married. Adm. Jan. 5. Disch. Jan. 28. In H. 23 days. Dr. Beale, vol. 5, p. 94. *Recovery.* First attack, 2 years ago. Prev. ill. 14 days. Pain in left side, palpitation and dyspnœa. On adm., knees inflamed (8 days), and afterwards left shoulder. Increased

cardiac dulness, systolic bruit over apex, intensified second sound. No pain in joints after Jan. 25.

Bicarbonate of potass and chloric ether, and quinine and opium.

Rheumatism—Acute.—T. H. W., æt. 35. Baker. Adm. Dec. 20, 1862. Disch. Feb. 4. In H. 46 days. Dr. Beale, vol. 5, p. 85. *Recovery* Teetotaller. Scarlet fever 7 yrs. ago, followed by dropsy and acute rheumatism. Prev. ill. 5 days. Shivering, headache and vomiting. Ankles and knees first affected, and then right wrist and elbow. On adm. soft systolic bruit over apex. On 12th day friction sound over heart (2 days), and on 13th, fine crepitus over right base. Highest pulse, 110: highest resp. 32.

Alkalies, quinine and opium. Afterwards with quinine and sulphuric acid.

*Erysipelas.—W. W., æt. 52. Malt worker. Adm. Dec. 24, 1862. Disch. Jan. 26. In H. 33 days. *Recovery.* Dr. Beale, vol. v, p. 80. Always very temperate. Prev. ill. 2 days. Rigors, followed by erysipelas of head and face. Urine normal.

Carbonate of ammonia, chloric ether and liquor cinchonæ, brandy up to 8 oz. Collodion and castor oil locally. Quinine and ext. of henbane during convalescence.

Scarlet Fever.—T. B., æt. 34. Butler. Adm. Dec. 22, 1862. Disch. Jan. 16. In H. 25 days. *Recovery.* Dr. Johnson, vol. 16, p 32. Prev. ill. 5 days. Sore throat and inflamed uvula. Desquamation on 10th day. Pain and tenderness of right shoulder on 14th day P. 80.

Quinine, chlorate of potass and dilute muriatic acid. Afterwards with quinine and muriated tincture of iron.

*Scarlet Fever and Typhoid Fever.—S. S., æt. 18. Servant. Adm. Jan. 21. Disch. March 16. In H. 54 days. *Recovery.* Dr. Beale, vol. 5, p. 112. Overworked. No contagion. Prev. ill. 4 days. Shivering, sore throat, great prostration. On adm., scarlet rash on face and arms, tonsils ulcerated, great dysphagia, tongue dry and brown. Delirium on 6th day. Two rose spots on abdomen on 7th day. Desquamation on 9th day, and 5 dark motions. On 11th day, jaws firmly clenched, cannot swallow. 12th day, swallows, and answers questions. No purging after 18th day. Convalescent on 31st day.

Liquor ammoniæ acetatis and chloric ether, and brandy 12 oz., increased to 18 oz. on 12th day. Injections of brandy, beef-tea, and quinine during trismus. Sulphate of copper and opium for the diarrhœa. During convalescence, dilute muriatic acid, chloric ether and pepsin.

Scarlet Fever.—M. A. W., æt. 17. Servant. Adm. Jan. 5. Disch. Jan. 31. In H. 31 days. *Recovery.* Dr. Beale, vol. 5, p. 55. Typhus, 5 years ago. Abscess in axilla, 10 days. No contagion. Prev. ill. 1 day. Sore throat, dysphagia, pain in back, vomiting. Eruption on 2nd day (4 days). Desquamation on 9th day. No ulceration of throat. Highest pulse, 140.

Liquor ammoniæ acetatis, and chloric ether, dilute muriatic acid during convalescence.

Scarlet Fever.—J. T., æt. 27. Book Mounter. Adm. Feb. 4. Disch. Feb. 28. In H. 24 days. *Recovery.* Dr. Johnson, vol. 15, p. 15. Probably caught from father. Prev. ill. 3 days, shivering, headache, nausea, sore throat, slight eruption on 2nd day. Tongue red and raw. P. 80. Desquamation on 11th day.

Chlorate of potass and muriated tincture of iron.

Febricula.—R. S., æt. 36. Housekeeper. Adm. Dec. 27, 1862. Disch. Jan. 10. In H. 14 days. *Recovery.* Dr. Johnson, vol. 16, p. 65. Febricula.

Quinine, tincture of sesquichloride of iron, and chloric ether.

Febricula.—E. M., æt. 20. Waistcoat Maker. Adm. Feb. 6. Disch. Feb. 21. In H. 15 days. *Recovery.* Dr. Johnson, vol. 16, p. 109. Febricula.

Liquor cinchonæ, nitro-muriatic acid and chloric ether.

*Typhoid Fever.—E. S., æt. 17. Servant. Adm. Feb. 5. *Died* on Feb. 8. Dr. Johnson, vol. 16, p. 103. No history of contagion. Prev. ill. 14 days. Shivering, diarrhœa 5 or 6 times in 24 hours, a *little* blood in motions, well marked and abundant rose spots, crepitus over bases of lungs, tympanites, unconsciousness, and death on 17th day. Highest pulse 148: highest resp. 40.—P. M. Ulceration of Peyer's patches and solitary glands of small intestine: no perforation. Spleen friable, wt. 14 oz. Lungs a little gorged.

Carbonate of ammonia, liq. cinch. and chloric ether; opium and sulphate of copper to check diarrhœa; brandy 12 *oz. increased to* 24 *oz. on* 16*th day.*

*Typhoid Fever.—C. T., æt. 10. Adm. Jan. 31. Disch. Feb. 25. In H. 25 days. *Recovery.* Dr. Beale, vol. 5, p. 115. Contagion. Prev. ill. 6 days. Rigors, headache, and lassitude. Abundant rose spots on abdomen on 7th day, no fresh ones after 9th day. Diarrhœa till 14th day, not more than four times in 24 hours, no blood in stools. Highest pulse 118; highest number of respirations 28.

Liquor ammoniæ acetatis, liquor cinchonæ and chloric ether, brandy 4 *oz., and stupes to chest. Quinine and tincture of sesquichloride of iron during convalescence.*

*Typhoid Fever.—H. C., æt. 30. Married. Adm. Feb. 11. *Died* on Feb. 18. Dr. Johnson, vol. 16, p. 115. Had been living badly before her illness contracted while in hospital. Prev. ill. 8 days. Shivering, headache, slight diarrhœa (16 days). Delirium on 5th day (11 days). On 10th day a few rose spots on trunk, tongue dry and brown. Death on 16th day. Highest pulse 140, highest resp. 44. P. M.—Intestines healthy: heart flabby and soft.

Carbonate of ammonia, liquor cinch, and chloric ether, brandy 8 *oz., increased to* 16 *on* 14*th day. Turpentine stupes to chest.*

*Typhus, Cardiac Disease.—H. C., æt. 22. Grocer's assistant. Adm. Nov. 26, 1862. Disch. Feb. 4. In H. 70 days. *Recovery.* Dr. Johnson, vol. 16, p. 11. Never had rheumatism. Pains in chest, shortness of breath and palpitation 5 years ago. Prev. ill. 3 days. Face livid, heart's impulse strong, thrill, dulness to right margin of sternum, scrobicular pulsation, double bruit, loudest at base, collapsing pulse. Crepitus over base of left lung. Urine normal. On Dec. 12th "caught cold." On Dec. 15th (1st day) headache and loss of appetite. On 7th day measly rash on trunk and arms, urine albuminous (till 13th day). On 11th day pulse 148, resp. 52. Delirium, slight diarrhœa (2 days). Rash gone on 12th day. Convalescent on 36th day.

Carbonate of ammonia and chloric ether on adm. During the fever, liquor cinchonæ, nitromuriatic acid and chloric ether, brandy up to 18 *oz., and turpentine stupes to chest. Tincture of sesquichloride of iron, and chloric ether during convalescence.*

*Typhus.—T. W., æt. 27, labourer. Adm. Feb. 18. Disch. Mar. 14. In H. 24 days. *Recovery.* Dr. Garrod, vol. 1, p. 52. No hist. of contagion or starvation. Prev. ill. 6 days. Rigors, headache, vomiting, pains in limbs, deafness. Cough on 3rd day. Delirium (10 days). Rusty expectoration, and indistinct rubeoloid eruption on 7th day. Highest pulse 128; highest resp. 48.

Nitromuriatic acid, liquor cinchonæ and chloric ether. Brandy 8 *oz., increased to* 12 *oz. on* 9*th day. Tr. opii m. x x. on* 9*th day. Turpentine stupes to chest.*

*Typhus,—W. E., æt. 22. Labourer. Adm, Jan. 3. Disch. Feb. 14. In H. 42 days. *Recovery.* Dr. Garrod, vol. 1, p. 4. Out of work 6 mos.; lived badly during that time. No contagion. Prev. ill. 14 days. No marked shivering.

Rubeoloid eruption first visible on Jan. 2. No traces of it left on Jan. 22. Urine albuminous till 4th day after adm. Delirium at night 9 days from admission. Highest pulse 144; highest resp. 40.

Liquor cinchonæ, nitro-muriatic acid and chloric ether; brandy 4 *oz., gradually increased to* 18 *oz. (on 9th day after ad.) Stupes to chest. Opium for delirium.*

*Typhus.—J. M., æt. 25. Orange woman. Adm. Feb. 18. Disch. Mar. 14. In H. 24 days. *Recovery.* Dr. Johnson, vol. 16, p. 121. No contagion. Prev. ill. 3 days. Shivering and headache. Rubeoloid eruption on 5th day. Tongue dry and brown. No delirium or diarrhœa. Highest pulse 120; highest resp. 36.

Liq. cinchonæ, nitro-muriatic acid and chloric ether, brandy 6 *oz.*

Typhus.—E. M., æt. 20. Servant. Adm. Dec. 13, 1862. Disch. Jan. 23. In H. 21 days. *Recovery.* Dr. Beale, vol. 5, p. 48. No history of contagion. Prev. ill. 14 days. Rigors, cough, headache, vomiting, distinct rubeoloid eruption. Highest pulse 124; highest number of resp. 46.

Liq. cinch. nitro-muriatic acid and chloric ether, brandy 4 *oz.*

*Typhus.—E. O'B., æt. 34. Laundress. Adm. Jan. 5. *Died* on Jan. 17. Dr. Garrod, vol. 1, page 8. No contagion.

Temperate, and usually lives well. Prev. ill. 7 days. After working 17 hours, attacked with shivering, headache, vomiting and diarrhœa. *Rose* eruption on 6th day. Marked *rubeoloid* eruption all over her on 11th day. Tongue dry and brown; delirium 5 days (from 11th day). Motions passed under her, and urine retained (12th, 13th, and 14th days). On 14th day harsh breathing over bases. On 16th day, rusty sputa: crepitus, and dulness over bases. Bronchial breathing over bases. Delirium. Death on 20th day. Highest pulse 140; Highest resp. 72. P. M.—Intestines healthy; lungs congested; brain not examined.

Brandy 6 *oz., gradually increased to* 24 *oz. Sulphate of copper to check dirrrhœa; liq. cinchonæ and aromatic spir. of ammonia; and turpentine stupes to chest* 4^{tis} *horis after* 14*th day.*

* Typhus.—G. S., æt. 18 Porter. Adm. Dec. 16, 1862. Disch. Jan. 24. In H. 39 days. *Recovery* Dr. Johnson, vol. 16, p. 29. Three other cases from same house. Prev. ill. 10 days. (?) Marked rubeoloid eruption, tongue dry and brown, active delirium for 5 days after adm. and albuminous urine 10 days. On 12th day P. 120, R. 40. Severe diarrhœa on 15th day.

Nitro-muriatic acid, liq. cinchonæ and chloric ether; brandy 6 *oz. increased to* 9 *oz. on* 15*th day. Muriated tincture of iron and chloric ether during convalescence.*

*Typhus.—E. C., æt. 72. Widow. Adm. Feb. 20. Disch. Mar. 18. In H. 26 days. Dr. Garrod, vol. 1, p. 58. *Recovery.* Slight attack of gout 12 years ago. Cough 7 years. Prev. ill. 3 days. Shivering, headache, vomiting, pains in limbs, large crepitus over bases, and viscid expectoration on 4th day. Measley rash over trunk on 5th day, tongue dry, no diarrhœa. Convalescent on 13th day.

Nitro-muriatic acid, liquor cinchonæ, and chloric ether, and brandy 6 *oz. Quinine and muriated tincture of iron during convalescence.*

*Pleurisy.—S. H., æt. 32. Servant. Adm. Nov. 8, 1862. Disch. Jan. 24. In H. 77 dys. *Recovery.* Dr. Johnson, vol 15, p. 29. Domestic servant, overworked. Amenorrhœa and debility (5 mon.) Prev. ill. 6 wks. with an attack of influenza (?), accompanied by severe pain in left side, and cough. Dulness on percussion up to angle of scapula on left side behind, and up to left mamma in front. Absence of respiratory murmur over dull region, and less expansion on that side. [Lateral curvature of spine to the right.] Systolic bruit over heart, loudest at apex. Dec. 10.—Dulness up to spine of left scapula. Dec. 24.

—Dulness very slight, feeble breathing heard all over base of lung. No friction sound audible at any time. Highest pulse 108, highest resp. 44.

Chloric ether, and tincture of sesquichloride of iron. Turpentine stupes and blisters.

*Pleurisy.—W. C., æt. 28. Tailor. Adm. Jan. 20. Disch. Feb. 9. In H. 20 day. *Recovery.* Dr. Johnson, vol. 15, p. 71. No family hist. of phth. Never had rheumatic fever. An attack of pleurisy (left side) 3 years ago (in K. C. H.), and 3 slighter pleuritic attacks since. Prev. ill. 9 days, with shivering and nausea; pain in left side (4 days) lasting till Feb. 1. Dulness up to angle of left scapula behind, and mamma in front; diminished vocal vibration, and no breathing over dull space; cough and yellowish viscid expectoration. On Jan. 30 (20th day), feeble respiration, murmur and crepitus (?) over base of left lung. No rub heard at any time. Highest pulse 100, highest resp. 28.

Carbonate of ammonia and chloric ether, and quinine and opium. During convalescence, quinine and tinct. of sesquichloride of iron.

*Pleurisy.—J. B., æt 30, Waiter. Adm. Oct. 4, 1862. Disch. Jan. 10. In H. 97 days. *Recovery.* Dr. Beale, vol. 6, p. 2. Drinks about 2 pots of beer and a quartern of spirits a-day. Lead Colic (13 weeks) 5 years ago. Prev. ill. 9 days, with shivering and pain in right side. On 8th day, dulness over whole of right side behind, and in front up to 4th rib (till discharged). Egophony at angle of right scapula (48 days). Rubbing over right lung (56 days). No albumen in urine.

Diuretics, and blisters to the affected side. Afterwards, with liquor cinch. and iodide of potassium, and then with tincture of sesquichloride of iron and bicarbonate of potass after meals.

*Acute Rheumatism—Pleurisy.—A. McD., æt. 16, Baker's boy. Adm. Jan. 21. Disch. Feb. 4. In hosp. 14 days. *Recovery.* Dr. Garrod, vol. 1, p. 26. Is much exposed to wet. On Dec. 11 (41 days before adm.) knees and ankles painful and swollen, and profuse sweating (8 days). On Jan. 1, knees and ankles again inflamed, and then wrists and elbows. On 21st day (day of adm.) increased cardiac dulness and systolic bruit over apex; pain in chest (8 days); dulness over left base, absence of breathing and vocal vibrations, bronchial breathing and œgophony at angle of left scapula. On 27th day, breathing over left base; friction sound (2 days). Highest pulse 94; highest No. of respns. 26.

Bicarbonate of potash and quinine for 5 days. Then the same with sulphate of iron gr. i for 8 days. Turpentine stupes to chest.

*Pneumonia.—W. C., æt. 35. Pressman. Adm. Jan. 20. Disch. Mar. 4. *Recovery.* Dr. Garrod, vol. 2, p. 12. Works in hot rooms. Intemperate of late. Prev. ill. 8 days. Shivering, headache, dyspnœa, pain in right side, cough, rusty viscid expectoration: delirium (6 days). On 9th day of disease, dulness (32 days) and bronchial breathing (16 days) over lower lobe of right lung; dulness and crepitus at base of left lung, only a trace of chlorides in urine. On 11th day, p. 116, r. 44. Tongue dry and brown; dulness and bronchial breathing under right clavicle and friction rub under right nipple.

Liquor ammonia acetatis, carbonate of ammonia and chloric ether; brandy up to 12 oz.; and turpentine stupes to chest. During convalescence, quinine and sulphuric acid, and syrup of iodide of iron.

*Pleuro-Pneumonia.—C. C., æt. 53. Adm. Mar. 4. *Died* on Mar. 5. Dr. Garrod, vol. 2, p. 63. No history. Semi comatose on adm. Breathing laborious, face livid, dulness over left side, back and front. No bronchial br. or bronchophony heard.

P.M. 0ii fluid in left pleura. The whole of left lung in stage of grey hepatization; recent lymph on its surface. Old adhesions of abdominal viscera.

Carbonate of ammonia and chloric ether, calomel purge, brandy, and turpentine stupes to chest.

*Pneumonia.—C. B., æt. 20. Groom. Adm. Jan. 6. Disch. Feb. 14. In H. 39 days. *Recovery.* Dr. Beale, vol. 6, page 58. Prev. ill. 4 days, after exposure to cold. On 5th day dulness behind up to spine of left scapula (31 days), and angle of right. Bronchial br. over these spaces, loudest on left side (17 days). Chlorides completely absent on 7th day. No expectoration. Highest number of respirations 40.

Liq. ammonia, acetatis and chloric ether, and afterwards with dilute muriatic acid. Blisters to left side. Brandy up to 6 oz.

*Pneumonia.—E. P., æt. 21. Railway telegraph clerk. Adm. Dec. 3, 1862. Disch. Jan. 17. In H. 45 days. *Recovery.* Dr. Johnson, vol. 15, p. 23. No exposure to wet or cold. Prev. ill. 10 days. Pain in left chest and shoulder. On adm. dulness to midway between spine and angle of left scapula, and as high as left nipple. Bronchial breathing, and bronchophony over same space. Expectoration not rusty.

Quinine, sulphuric acid and tincture of sesquichloride of iron and cod-liver oil. Turpentine stupes, and Ung. Iodinii co. to left side.

Readmitted on Jan. 21, for quinsy. Discharged cured on Jan. 28. Treated with carbonate ammonia and chloric ether, and afterwards with quinine and tincture of sesquichloride of iron.

*Pneumonia.—T. H., æt, 24. Upholsterer's porter. Adm. Feb. 11. Disch. Feb 28. In H 17 days. *Recovery.* Dr. Johnson, vol. 16, p. 117. Exposed to cold while sweating. Prev. ill. 5 days. Headache, pain in left side and dyspnœa. On 5th day, dulness and crep over lower third of left lower lobe (till 12th day). Expectoration streaked with blood: total absence of chlorides from urine (1 day). P. 116, R. 36. Convalescent on 13th day.

Liq. ammon. acetat. and chloric ether, and turpentine stupes. Quinine, sulphuric acid and chloric ether during convalescence.

*Pneumonia.—C. L., æt 19. Servant. Adm. Mar. 2. Disch. Mar. 21. Twenty days in H. *Recovery.* Dr. Johnson, vol. 15, p. 119. Has had two attacks of rheumatic fever. On Feb. 20 "caught cold." On Feb. 27th (1st day) rigors, pain in right side and limbs, headache, rusty, viscid expectoration (7 days). On 3rd day, dulness up to angle of right scap., bronch. breathing and bronchophony, tongue furred: chlorides of urine much diminished. Returning crepitation on 6th day. Very little crepitation remaining on 10th day.

Liquor ammon. acetat. and chloric ether, 6 oz., brandy and stupes to chest night and morning. Quinine and sulphuric acid during convalescence.

*Pneumonia.—T. W., æt. 27. Carver and gilder. Adm. Mar. 4. Disch. Mar. 28. In H. 24 days. *Recovery.* Dr. Johnson, vol. 15, p. 121. Is exposed to great variations of temperature. Eight days before ad. "caught cold." Six days before ad. shivering, headache, cough, dyspnœa, viscid, yellow sputa (15 days); pain below rt. nipple. On 7th day, face dusky, herpes on lower lip, dulness up to spine of right scapula, bronchial breathing and bronchophony; only a trace of chlorides in urine. Returning crep. on 7th day. Rubbing over rt. lateral region on 22nd day (8 days). Convalescent on 30th day.

Liq. ammon. acetat. carbonate of ammonia and chloric ether. Turpentine stupes. Afterwards with quinine and sulphuric acid.

*Pneumonia.—N. W., æt. 46. Commissionaire. Adm. Dec. 30, 1862. Disch. Jan. 24. In H. 25 days. *Recovery.* Dr. Johnson, vol. 16, p. 56. In army 21 yrs. Prev. ill. 6 days. Pain in right side, cough, dyspnœa. On 6th day dulness up to spine of right scapula, fine crepitation, and bronchophony over same space. Rhonchus all over chest, sputa rusty (10 days). On 7th day p. 116, r. 40. Tongue dry and brown, sputa very rusty and viscid. Convalescent on 16th day.

Liq. ammon. acetat. chloric ether and antimonial wine, brandy up to 8 oz. Turpentine stupes. Afterwards quinine, sulphuric acid and chloric ether.

*Pneumonia.—L. E., æt. 12. Adm. Feb. 3. Disch. Feb. 21. In H. 18 days. *Recovery.* Dr. Johnson, vol. 16, p. 97. Always delicate. Had measles

5 wks. ago. Prev. ill. 1 wk.; shivering, cough and expectoration. On 7th day of disease fine crepitation, and dulness over both bases. P. 136, r. 48. Expect. scanty, viscid: not rusty. No crepitation heard after 15th day.

Liq. ammon. acetatis carbonate of ammonia and chloric ether; a purge; and turpentine stupes.

*Pneumonia.—R. H., æt. 22. Servant. Adm. Jan. 17. *Died* on Jan. 19. Dr. Johnson, vol. 15, p. 49. Prev. ill. 6 days. Shivering and headache. Pain in right side (5 days). Delirium on 6th, 7th and 8th days. When admitted (7th day) was bleeding profusely from leech bites, and very weak. Dulness over *upper* lobe of right lung, bronchial breathing in front, harsh breathing and a rub behind. P. 140, r. 80. Death on 8th day of illness. P. M. Upper lobe of right lung being hepatized; recent lymph on its surface; remainder of lungs engorged.

Carbonate of ammonia, liquor ammoniæ, acetatis, and chloric ether; brandy increased from 6 oz. up to 24 oz. Turpentine stupes.

*Pneumonia.—W. B., æt. 37. Commissionaire. Adm. Dec. 6, 1862. Disch. Jan. 17. In H. 48 days. *Recovery.* Dr. Beale, vol. 5, p. 64. While in India had two attacks resembling dysentery. Much exposed to wet and cold. Prev. ill. 6 days. Shivering, cough and dyspnœa. On 10th day from shivering, fine crepitation and dulness over right lower lobe; urine slightly albuminous; much chloride. On 11th, 12th, and 13th days active delirium; chloride absent from urine. Urine and fæces passed involuntarily. On 13th day bronchial breathing over right base; by 16th day this extended up to spine of right scapula, and 3rd rib in front; dulness over this region. P. 120, r. 56. Tongue dry and brown: face livid. Returning crepitation on 17th day, and rusty sputa. Bronchial breathing audible for 29 days, and dulness for 43.

Liq. ammoniæ acetatis, carbonate of ammonia, and chloric ether; calomel purges; tr. opii. mxv 2ndis *horis during delirium, and cupping to 5 oz. over back. Afterwards liquor cinchonæ, nitro-muriatic acid, and mag. sulphat. Brandy 6 oz. afterwards increased to 12 oz.*

*Pneumonia.—A. P., æt. 24. Married. Adm. Dec. 19, 1862. Disch. Jan. 24. In H. 36 days. *Recovery.* Dr. Beale, vol. 5, p. 74. Lives in a damp house. Prev. ill. 6 days. Rigors; cough; dyspnœa; pain under right breast; yellow expectoration. On 7th day, dulness up to spine of right scapula. Bronchial breathing, bronchophony and crepitus over same space. Rhonchus in front. On 10th day (of disease) dulness and bronchial breathing under right clav. over a space of 3 in. diam.

Liq. ammonia acetatis, carbonate of ammonia and chloric ether, and afterwards tincture of sesquichloride of iron, and cod-liver oil. Turpentine stupes to right side, and counter-irritation under right clavicle by a small blister.

*Acute Phthisis.—M. A. G., æt. 31. Prostitute. Adm. Jan. 30. *Died* on Feb. 7. In H. 8 days. Dr. Garrod, vol. 1, p. 34. Very intemperate. Prev. ill. 36 days, with cough, great emaciation, and loss of strength. Delirium, sleeplessness and constipation, 8 days. On adm. face flushed, tongue dry, evacuations passed under her. P. 134, r. 36. Dulness and large crepitation over right apex, and to a less extent over left apex. Sputum viscid, and tinged with blood. No *p. m.* allowed.

Carbonate of ammonia and chloric ether, and brandy 7 oz, increased to 12 oz. on 8th day after adm.

Phthisis.—S. S., æt. 30. Housemaid. Adm. Jan. 28. Disch. Feb. 23. In H. 26 days. *Relieved.* Dr. Johnson, vol. 16, p. 91. Pains in chest, emaciation, and debility, 2 yrs. Vomiting of dark blood on two occasions (10 wks.). Catamenia irregular. Dulness over right apex, and crepitus and harsh breathing under clavicles. Purulent expectoration. P. 98. No hœmopt. or night sweats. Very anœmic.

Sesquichloride of iron, and cod-liver oil.

Phthisis.—W. P., æt. 40. Carpenter. Adm. Jan. 26. Disch. Feb. 7. In H.

12 days. *Slightly relieved.* Dr. Johnson, vol. 15, p. 77. Intemperate. No hereditary predisp. Cough and expectoration 5 yrs. Slight hœmoptysis 2½ yrs. Night sweats. Not much emaciation. Large crepitation and dulness over whole of left lung behind, crepitation over base of rt. lung; wooden resonance over front of left lung, where bronch. breathing, amphoric blowing, and metallic tinkling. Copious, purulent expectoration, containing lung tissue.

Sesquichloride of iron, cod-liver oil, and chlorodyne.

PHTHISIS.—M. A. L., æt. 64. Married. Adm. Jan. 20. Disch. Feb. 4. In H. 15 days. *Relieved.* Dr. Beale, vol. 51, p. 99. Lives in a damp house. Two brothers died of phth. Prev. ill. 2 yrs. Palpitation, shortness of breath, debility and loss of flesh, cough, and hœmoptysis (to about 3 oz.) 7 or 8 times a year. Pain, dulness and bronchial breathing under right clav. Basic systolic bruit, and increased intensity of 2nd sound. Slight loss of power in right arm with tingling of 3rd and 4th fingers.

Sulphuric acid and digitalis.

PHTHISIS.—A. C., æt 40. Needlewoman. Adm. Jan. 7. Disch. Jan. 31. In H. 24 days. *Relieved.* Dr. Beale, vol. 6, p. 66. No hereditary predisp. Slight hœmoptysis 3 yrs. ago. Prev. ill. 3 mos. Exposure to cold followed by cough and expectoration, and dyspnœa, loss of strength and swelling of feet and ankles. Pain, large crep. and dulness under left clavicle.

Tincture of sesquichloride of iron and henbane.

LARYNGEAL PHTHISIS.—R. C., æt. 30. Labourer. Adm. Jan. 7. Disch. Mar. 14. In H. 38 days. *Relieved.* Dr. Johnson, vol. 15, p. 55. Exposed to great variations of temperature. A severe cold 10 mos. ago, followed by gradual loss of voice and strength. On adm. speaks in a hoarse whisper. Epiglottis red, thickened and granular, false cords swollen and vascular. Dulness and prolonged exp. under rt. clavicle. Expectoration profuse; viscid mucus streaked with blood. On Feb. 13, dulness and crepitus over rt. apex behind and in front; voice improved.

Cod liver oil, quinine, creosote inhalation, and blister over larynx.

LARYNGEAL PHTHISIS.—C. G., æt. 32. Clock-maker. Adm. Feb. 12. Disch. at own request Mar. 4. In H. 20 days. *Not improved.* Dr. Johnson, vol. 15, p. 93. No hereditary predisp. Pleurisy (rt. side) 3 yrs. ago. Cough and expectoration 3 yrs. About 18 mos. ago, slight dysphagia and gradual loss of voice. On adm. a deep ulcer ½ an inch long on left border of epiglottis, which was much thickened. Prolonged expiration under rt. clavicle.

Quinine, acid, and glycerine, cod-liver oil, creosote inhalation, and local application of sd. A.N.

CHRONIC BRONCHITIS.—L. McN., æt. 49. Cook. Adm. Nov. 15, 1862. Disch. Jan. 17. *Relieved.* Dr. Beale, vol. 6, p. 33. Two attacks of scarlatina, at 7 and 23 yrs. of age. Winter-cough 12 yrs. Prev. ill. 3 wks. with cough and dyspnœa. Afterwards rheumatic pains in knees and shoulders.

Carbonate of ammonia, chloric ether and senega, and turpentine stupes.

CHRONIC BRONCHITIS.—M. C., æt. 60. Laundress. Adm. Dec. 9, 1862. Disch. Jan. 17. In H. 39 days. *Recovery.* Dr. Beale, vol. 6, p. 41. Not subject to winter-cough. Prev. ill. 1 mon. Cough, dyspnœa, and frothy expectoration. Rhonchus all over chest on adm. Heart healthy. No albumen.

Ammonia, chloric ether, and liquor cinchonæ, and turpentine stupes. Afterwards quinine and sulphuric acid.

BRONCHITIS.—Eliz. Lawrence, æt. 51. Married. Adm. Jan. 15. Disch. Mar. 14. In H. 58 days. *Recovery.* Dr. Garrod, vol. 2, p. 1. Brother died of phth. Winter-cough 3 years. Typhus 8 mon. ago; ailing since. Prev. ill. 2 mon. Cough and expectoration; dry sounds, chiefly over right chest; no dulness. P. 108, R. 36.

Carbonate of ammonia, chloric ether and senega, turpentine stupes and brandy 4 oz. Afterwards quinine and sulphate of iron.

Bronchitis.—M. A. W., æt. 45. Servant. Adm. Dec. 31, 1862. Disch. Jan. 21. In H. 21 days. *Recovery.* Dr. Beale, vol. 5, p. 87. Acute rheumatism 13 yrs. ago. Winter-cough 3 yrs. Prev. ill. 3 days. General bronchitis following exposure to wet and cold. Heart's sounds normal.

Carbonate of ammonia, chloric ether and senega, and turpentine stupes. Afterwards quinine and sulphuric acid.

*Bronchitis.—W. P., æt. 38. Labourer. Adm. Mar. 15. *Died* on Mar. 17. Dr. Beale, vol. 5, p. 142. Acute bronchitis 8 years ago. Prev. ill 8 days, commencing with rigors, cough and pain in chest. On adm. cough and dyspnœa, scanty expectoration, rhonchus and sibilus all over lungs. Lips blue. P. 104, R 40. Delirium on 16th. P.M.—Lungs emphysematous. Bronchial mucous membrane inflamed, air-passages choked with mucus.

A calomel purge, and carbonate of ammonia, and chloric ether; brandy 8 oz., increased to 12 oz. on 16th. V. S ad ʒviij on 15th, and again ad ʒiv. Turpentine stupes to chest.

*Bronchitis.—G. R., æt. 44. Formerly hospital porter. Transferred from surgical ward Oct. 18, 1862. Disch. Jan. 15. *Recovery.* In H. 89 days. Dr. Johnson vol. 14, p. 245. Intemperate. In army for 25 years. Amputation of left hand on Sept. 20, for disease of wrist-joint, was followed by erysipelas of arm. During convalescence pain in right side (1 wk. before adm.) Cough and frothy expectoration, dyspnœa, tongue dry and brown. P. 126, r. 36. Rhonchus all over chest. No history of tubercle.

Nitric acid, liquor cinchonæ and chloric ether; turpentine stupes, and brandy up to 12 oz.

Emphysema and Bronchitis.—Laura P., æt. 48. Adm. Feb. 20. Disch. Mar. 21. In H. 29 days. *Recovery.* Dr. Beale, vol. 6, p. 100 Acute rheum. 30 yrs. ago. "Brain fever" 28 yrs. ago. Occasional swelling of legs 12 yrs. Winter cough 2 yrs. On adm. cough, and viscid greenish expectoration; chest barrel-shaped and resonant, rhonchus all over it; scrobicular pulsation; no bruit. P. 94.

Carbonate of ammonia and chloric ether, and afterwards nitric acid and bark.

*Emphysema and Bronchitis – Gout.—C. I, æt., 51. Carpenter. Adm. Jan. 10. Disch. Mar. 4. In H. 53 days. *Relieved.* Dr. Beale, vol. 6, p. 62. Much exposed to wet and cold. Moderately temperate. Forty attacks of gout in last 12 yrs. Winter cough 12 yrs. Two years ago, dropsy of legs (4 mos.). Prev. ill. 12 wks., with cough and dyspnœa. Dry sounds over lungs. No albumen in urine, but granular casts. An attack of general gout on 5th day after adm., commencing in left knee. Dropsy of legs on 37th day after adm.

Carbonate of ammonia and chloric ether, and turpentine stupes. Then with iodide of potassium, and bicarbonate of potass, and afterwards tincture of sesquichloride of iron. Blisters above ankles and left wrist.

Emphysema and Bronchitis.—M. C., æt. 48. Needlewoman. Adm. Jan. 3. Disch. Jan. 17. In H. 14 days. *Recovery.* Dr. Johnson, vol. 15, p 53. Winter cough, shortness of breath, and occasional swelling of legs 5 yrs. Face livid; chest barrel-shaped; pulsation in scrobiculus; no bruit; urine albuminous.

Carbonate of ammonia, chloric ether, and senega, and turpentine stupes.

*Emphysema and Bronchitis.—T. W., æt. 32. Chimney sweep. Adm. Nov. 21, 1862. *Died* on Jan. 25. In H. 65 days. Dr. Budd, vol. 74, p. 110. Intemperate. Never had rheum. Cough 5 yrs. Pain in loins 3 yrs. Slight hœmoptysis 1 yr. Prev. ill. 14 days. Face livid. Legs and belly dropsical. Purpuric eruption all over body. Systolic bruit, loudest in scrobiculus. Urine very albuminous for 11 days after adm. Inflammation of left leg on Jan. 24. P. 120, R. 46.

P.M. Large bladders on edges of lungs; small cavity in lower lobe of left. Wt. of heart, 12 oz.; right ventricle dilated and hypertrophied; valves healthy. Serous fluid in abdomen.

*Emphysema and Bronchitis.—R. W., æt. 54. Potman. Adm. Mar. 14. *Died* on Mar. 29. In H. 15 days. Dr. Johnson, vol. 15, p. 133. Intemperate. Winter cough 11 yrs. Prev. ill. 3 mos. On adm. face dusky; dyspnœa, cough, and viscid expectoration; chest barrel-shaped, and unduly resonant; rhonchus and sibilus heard behind; heart's sounds normal. P. 104, r. 36.

P.M.—Recent pericarditis. Wt. of heart 20½ oz.; left ventricle much hypertrophied; tricuspid orifice and right vent. dilated; lungs emphysematous. Kidneys in early stage of granular degeneration.

Carbonate of ammonia, chloric ether, and bicarbonate of potass. Afterwards with quinine and chloric ether, and emetics. Brandy 4 oz., gradually increased to 12 oz. Turpentine stupes to chest.

*Acute Laryngitis.—J. W., æt. 47. Porter. Adm. Feb, 12. *Died* Feb. 17. In H. 9 days. Dr. Beale, vol. 6, p. 88. Intemperate and much exposed to wet and cold. Had Syphilis. Prev. ill 7 days, with sore throat and dysphagia. Œdema of legs 4 days. Aphonia and stridulous breathing for 2 days before adm. Rhonchus over lungs. Trace of albumen in urine. Laryngoscope showed swelling, redness of superior vocal cords, immobility of lower cords. Tracheotomy on fourth day after adm. *Death* five days after operation.

P.M.—Vocal cords as above. A cavity containing shells of bone in the situation of each arytenoid cartilage. Ossification of thyroid and cricoid, and rings of trachea. Bronchial mucous mem. considerably inflamed.

Conium and ipecacuanha 1 day, and then with liquor ammoniæ acetatis, carbonate of ammonia, and chloric ether. Brandy up to 12 oz., and steam inhalation.

*Croup.—J. D., æt. 4. Adm. Jan. 9. Disch. Jan. 28. In H. 19 days. *Recovery.* Dr. Johnson, vol. 16, p. 72. Delicate and subject to coughs. Cough (10 days), followed two days bef. adm. by aphonia and croupal breathing (3 days). P. 136, r. 40. Rhonchus over chest. Convalescence. On 6th day after adm. a second attack of about 50 hours' duration.

An ipecacuanha emetic, calomel and jalap purge, and carbonate of ammonia and chloric ether. Steam inhalation.

Croup.—F. B., æt. 3½ yrs. Adm. Feb. 4. Disch. Feb. 17. In H. 13 days. *Recovery.* Dr. Beale, not in case book. On adm. cough, hoarseness, slight dyspnœa, no strid, pulse rapid.

An ipecacuanha emetic, liquor ammon. acetatis, ipecac. and chloric ether, wine 2 oz., steam inhalation and external application of belladonna.

Cardiac Disease.—J. C., æt. 28. Labourer. Adm. Jan. 17. Disch. Feb. 9, In H. 23 days. *Relieved.* Dr. Johnson, vol. 16, p. 77. No rheumatism. Prev. ill. 1 yr. Pain in chest, palpitation, and shortness of breath. Heaving impulse, thrill over sternum, cardiac dulness twice the natural extent. Musical diastolic bruit, loudest at base. Pulse 60, collapsing. No dropsy.

Quinine, tincture of sesquichloride of iron, and chloric ether.

Cardiac Dropsy.—Maria C., æt. 42. Adm. Jan. 17. Disch. Feb. 28. In H. 42 days. *Relieved.* Dr. Beale, vol. 6, p. 70. Winter-cough 6 yrs. Prev. ill. 1 mon. Dropsy of feet and ankles. Dyspnœa, cough, and frothy expectoration. Ascites. Systolic bruit over base of heart. Rhonchus and large crepitation over lungs. Urine very albuminous.

Liquor ammoniæ acetatis, carbonate of ammonia and chloric ether. Henbane and conium pills at bedtime, and turpentine stupes.

*Cardiac Dropsy.—Eliz, G., æt. 38, Married. Adm. Feb. 2. *Died* Feb. 15.

In H. 13 days. Dr. Garrod, vol. 1, p. 39. No rheumatism, or family hist. of phthisis. Cough and occasional hœmoptysis 4 yrs. Diarrhœa 3 wks, followed by dropsy of legs and ascites. Dulness and crepitus at right apex, and slight dulness at right base. Viscid expectoration. Systolic bruit at apex of heart. Urine $\frac{1}{5}$th albumen. Severe pain and tenderness of abdomen shortly before death.

P.M.—Lungs emphysematous, bronchial mucous membrane inflamed, scattered tubercles in lungs. Fluid in pleuræ. Right ventricle dilated, tricuspid orifice admitted 4 fingers. Sero-purulent fluid in abdomen. Recent lymph on intestines. Kidneys fatty.

Carbonate of ammonia and chloric ether, jalap and cream of tartar, and cod-liver oil.

*Cardiac Dropsy.—A. B., æt. 47. Tailor. Adm. Feb. 5. *Died* on Feb. 15. In H. 10 days. Dr. Beale, vol. 5, p. 118. Acute rheum. 14 yrs. ago. Palpitation, shortness of breath, and cough 4 yrs. Hœmoptysis, ℥ss, 16 mon. ago. Scrobicular pulsation, 15 mon. Loss of flesh 2 mon. Pain in left side 14 days. Dropsy of feet and ankles 7 days. Extremely anœmic. Double bruit over heart, loudest at base. Collapsing pulse. Urine very scanty and albuminous. Died rather suddenly.

P.M.—Wt. of heart, 1$\frac{3}{4}$lb. Coronary veins enlarged. Aortic and mitral valves thickened, and dilatation of arch of aorta. Recent lymph on intestines.

Carbonate of ammonia, and chloric ether, and ammonio-citrate of iron, and compound jalap powder.

*Cardiac Dropsy.—S. B., æt. 19. Shopman. Adm. Mar. 4. Disch. at own request, Mar. 21. In H. 17 days. Dr. Beale, vol. 6, p. 108. Acute rheumatism, accompanied by pain over heart, and delirium 7 yrs. ago. Several attacks since. Cough 3 yrs. Prev. ill. 2 mon. Frequent fainting-fits. On adm. face and lips blue, breathing hurried, œdema of legs, heart's action very irregular. Cardiac dulness from 2 in. to left of left nipple to right edge of sternum, systolic bruit over apex. Liver enlarged and tender. No albumen in urine. Jaundice on 4th day after adm. Vomiting and purging on 8th and 9th days.

Ammonia, chloric ether and opium, and a purge. Vomiting checked by hydrocyanic acid and soda, and diarrhœa by opiate enemata.

*Cardiac Dropsy.—Eliza B., æt. 50. Married. Adm. Dec. 23, 1862. Disch. Jan. 10. *Relieved.* Re-admitted Jan. 30. *Died* on Feb. 7. Dr. Johnson, vol. 15, p. 47. Acute rheumatism 14 yrs. ago, when pain over heart and delirium. Prev. ill. 12 mon. Palpitation, dyspnœa, cough, hœmoptysis, and dropsy of legs, very great on admission. Face livid, great distress of breathing, pulse small and irregular, 128, loud systolic bruit over apex of heart, crepitus over bases of lungs. Urine $\frac{1}{2}$ albumen (none after 15th day). Treated with diuretics and stimulants.

After her discharge the dropsy returned, and the breathing became more and more embarrassed. On Jan. 30 sudden left hemiplegia with temporary loss of consciousness, sensation slightly impaired, deglutition difficult, left arm and leg more swollen than right. P. 106, irregular; albumen $\frac{1}{4}$th. Urgent dyspnœa on Feb. 5th, going off after slight hœmoptysis. Fine crepitation at angle of right scapula on Feb. 6th.

P.M.—Right corpus striatum softened, and containing large granular cells, middle cerebral art. pervious. Patches of pulmonary apoplexy in lower lobes. Great thickening of mitral valve, pulmonary artery dilated. Kidneys granular, and studded with cysts.

Enemata of beef-tea and quinine, brandy 8 *oz., ammonia and ether, and quinine and chloric ether.*

*Cardiac Dropsy.—Mary A. B., æt. 27. Married. Adm. Jan. 16. *Died* on Feb. 15. In H. 30 days. Dr. Johnson, vol. 16, p. 63. Acute rheumatism 4 yrs. ago. Shortness of breath 15 months. Prev. ill. 9 mon. Nausea and vomiting, swelling of legs followed by ascites. Systolic bruit over apex of heart. No albumen in urine. Moist sounds over bases on 3rd day after adm. Legs punctured on 17th day after adm. Drain of fluid from punctures afforded great relief for a time. On 28th day after adm. right leg inflamed; slight jaundice.

P.M.—Wt. of heart 16 oz., auricles dilated, left ventricle hypertrophied. Mitral valve very thick, its edges cartilaginous.

Carbonate of ammonia and chloric ether on adm., then with diuretics and elaterium. Brandy and wine up to 8 oz.

Inflammation of knee-joint.—E. B., æt. 23. Chandler. Adm. Dec. 15, 1862. Disch. Jan. 21. In H. 9 days. *Recovery.* Dr. Beale, vol. 5, p. 88, Strumous. Not very temperate. Pain, tenderness, and swelling of right knee, following a long walk. P. 116. No Pain after 16th day.

Chiefly, iodide of potassium, colchicum, and bicarb. of potass, and afterwards with ammonio-citrate of iron, and cod-liver oil.

Subacute Rheumatism.—T. H., æt. 35. Hospital Porter. Adm. Feb. 19, Disch. Feb. 28. In H. 9 days. *Recovery.* Dr. Garrod, not in case book. Very mild attack. No heart affection.

Iodide of potassium, and bicarbonate of potass.

Subacute Rheumatism.—L. E., æt. 28. Cook. Adm. Feb. 18. Disch. Mar. 6. In H. 16 days. *Recovery.* Dr. Beale, vol. 6, p. 96. Acute Rheumatism 6 yrs. ago (5 wks.) Prev. ill. 3 days. Feet and ankles painful and swollen. Moderate sweating. Basic systolic bruit over heart on adm.

Bicarbonate of potass, quinine and opium.

Subacute Rheumatism.—S. G., æt. 20. Servant. Adm. Feb. 19. Disch. Mar. 4. In H. 13 days. *Recovery.* Dr. Beale, vol. 5, p. 129. First attack. Prev. ill. 4 days. Left wrist and right shoulder affected. No Cardiac disease.

Liquor ammoniæ acetatis and chloric ether, and purgatives.

Subacute Rheumatism & Chronic Bronchitis.—A. D., æt. 68. Orange-woman. Admitted Dec. 10, 1862. Disch. Feb. 7. In H. 59 days. *Relieved.* Dr. Beale, vol. 6, p. 45. Acute rheumatism 21 yrs. ago. Since then has lived badly, and been much exposed to wet and cold. Cough 6 months. Prev. ill. 1 month, with pain and swelling of right ankle. On adm. œdema of legs and feet. P. 76. Left shoulder afterwards affected. No cardiac disease.

Liquor ammoniæ acetatis, chloric ether, bicarbonate of potass, and decoction of senega. Afterwards with muriated tincture of iron. Blisters to left shoulder.

Subacute Rheumatism.—J. G., æt. 27. Gardener's Labourer. Adm. Feb. 13. Disch. Feb. 25. In H. 12 days. *Recovery.* Dr. Garrod, vol. i, p. 50. Two former attacks 9 yrs. and 18 mon. ago. Shortness of breath and cough 18 mon. Prev. ill. 14 days. No exposure to wet. Commenced in left ankle; wrists, elbows, shoulders and knees attacked about 11th day. Diastolic bruit over base of heart. Slight sweating. Excess of urea in urine. P. 82.

Quinine and bicarbonate of potass.

Subacute Rheumatism.—W. L., æt. 30. Labourer. Adm. Feb. 16. Disch. March 11. In H. 23 days. *Recovery.* Dr. Beale, vol. 6, p. 84. First attack 11 yrs. and second 4 yrs. ago. Prev. ill 7 days. Ankles and knees first attacked, and then wrists. Systolic bruit over apex of heart on 15th day.

Bicarbonate of potass, quinine, and opium.

Subacute Rheumatism.—E. O. Cook, æt. 19. Adm. Feb. 11. Disch. Feb. 25. In H. 14 days. *Recovery.* Dr. Garrod, vol. 2, p. 38. Hard worked. Scarlet fever 5 years ago. followed by acute rheumatism; no carditis. Prev. ill. 11 days, with præcordial pain and dyspnœa. On 4th day pain and swelling of left ankle, and acid sweats, followed by pain in knees and hips. P. 84. Systolic bruit over base of heart; venous murmur in neck. Convalescent on 21st day.

Bicarbonate of potass (3ss doses), and quinine for half the time, and then with quinine, sulphate of iron, and sulphuric acid.

Subacute Rheumatism.—W. J., æt. 21. Pugilist. Adm. Feb. 14. Disch. Mar. 4. In H. 18 days. *Recovery.* Dr. Johnson, vol. 15, p. 97. A hard drinker.

Pains in limbs 4 mon. after sleeping on grass for 5 nights. Prev. ill. 18 days. Knees, ankles, hips, shoulders, elbows, wrists, affected in succession. Cough and profuse expectoration. Pain and increased dulness over heart. Basic systolic bruit.

Bicarbonate of potass, quinine, and opium, and afterwards with carbonate of ammonia, and ammonio-citrate of iron.

Subacute Rheumatism.—Cath. P., æt. 46. Married. Adm. Nov. 25, 1862. Disch. Jan. 17. In H. 53 days. *Recovery.* Dr. Budd, vol. 73, p. 100. No family history of rheumatism or gout. Drinks spirits. Slight attack of rheum. 3 yrs. ago. Prev. ill. 2 days. Knees and ankles first affected, afterwards wrists and shoulders. A little pain in left shoulder on Jan. 9. Moderate sweating. No cardiac disease. Highest pulse 130.

Bicarbonate and nitrate of potass, and colchicum and opium. Afterwards with bicarbonate of potass and quinine.

Acute Rheumatism & Bronchitis.—A. B., æt. 32. Needlewoman. Adm. Dec. 22, 1862. Disch. Jan. 31. In H. 40 days. *Recovery.* Dr. Johnson, vol. 16, p. 46. Acute rheumatism 15 yrs. ago. Shortness of breath 5 yrs. Bronchitis and dropsy of feet and ankles 3 yrs. ago. On adm. joints of right side inflamed. Systolic bruit over apex. On 18th day after adm. double bruit over base. Moist sounds over lungs. Highest pulse 104: highest resp. 22.

Bicarbonate and nitrate of potass, chloric ether and ammonio-citrate of iron. Turpentine stupes to chest.

*Acute Rheumatism.—S. A. de K., æt. 12. Adm. Jan. 22. Disch. Mar. 21. In H. 58 days. *Recovery.* Dr. Johnson, vol. 15, p. 75. Very anœmic. No exposure to cold. Prev. ill. 5 days. Commenced in left ankle, then in left knee, right ankle, right knee, hips, shoulders, elbows and wrists. P. 100. Cardiac dulness slightly increased. Basic systolic bruit. On 25th day double bruit at base. Pulse collapsing. Convalescent on 40th day.

Bicarbonate of potass, quinine and opium. Quinine and sulphate of iron during convalescence. Blister to right shoulder.

*Acute Rheumatism.—E. G., æt. 22. Servant. Adm. Dec. 16, 1862. Disch. Jan. 31. In H. 60 days. *Recovery.* Dr. Johnson, vol. 16, p. 27. Cough and occasional hœmoptysis, 3 yrs. Catamenia irregular. Prev. ill. 14 days, after getting wet through on two successive days. Ankles, knees, elbows, and shoulders affected. Systolic bruit loudest over base. Profuse sweating. Convalescent on 30th day. Highest pulse 124: highest resp. 48.

Bicarbonate of potash, and quinine, and opium, and ammonio-citrate of iron.

Acute Rheumatism.—John M., æt. 13. No occ. Adm. Feb. 5. Disch. Feb. 18. In H. 13 days. *Recovery.* Dr. Garrod, vol. 1, p. 42. Strumous disease of hip, 6 yrs. Prev. ill. 7 days. Pain and swelling of left knee and right wrist, afterwards of right elbow. Pain over heart till 8th day. Systolic bruit over apex on adm. Profuse sweating till 14th day. Highest pulse 112.

Bicarbonate of potass and quinine.

*Acute Rheumatism.—H. B., æt. 17. Nursemaid. Adm. Jan. 15. Disch. Mar. 14. In H. 59 days. *Recovery.* Dr. Garrod, vol. 2, p. 4. Prev. health good. First attack. Prev. ill. 14 days. Redness, pain, and swelling of left shoulder, after getting wet; then of wrists, hips, knees, and ankles. Profuse acid sweats. On adm. knees and right ankle inflamed. Knees well on 19th day, ankle on 54th day. No cardiac disease.

Alkalies and quinine for 16 days. Then iodide of potassium for 24 days, and syrup of iodide of iron during convalescence.

*Acute Rheumatism.—G. F., æt. 55. Coach-driver. Adm. Jan. 31. Disch. Mar. 14. In H. 42 days. *Recovery.* Dr. Garrod, vol. 1, p. 37. Intemperate. Thirty years ago general dropsy, after exposure to cold. Prev. ill. 3 wks. Began in ball of left great toe, then ankles, knees, shoulders, elbows, wrists, and small

joints of hand. Profuse sweating. P. 108. No family history of gout. No excess of uric acid in blood serum. No pain remaining on 55th day.

Bicarbonate of potass (℈ii) *and quinine. Then with carbonate of ammonia* (10 *gr. doses*), *and quinine, and decoction of bark. Brandy* 6 *oz. afterwards changed for gin.*

Rheumatism.—W. L., æt. 33. Compositor. Adm. Dec. 2, 1862. Disch. Jan. 26. In H. 55 days. *Much relieved.* Dr. Budd, vol. 74, p. 119. Inflammation of ball of right great toe during last 3 winters. Gonorrhœa 1 month. Prev. ill. 3 wks. with inflammation of ball of right great toe 37 days, ankles (76 days), and small joints of hand (20 days). Heart unaffected. Still some pain in ankles on discharge.

Principally, bicarbonate and iodide of potassium, and decoction of bark. Afterwards with guiacum, large doses of iodide of potass, and quinine, and acid.

*Sciatica.—J. C., æt. 48. Farrier. Adm. Dec. 12, 1862. Disch. Jan. 28. In H. 48 days. *Recovery.* Dr. Budd, vol. 73, p. 116. Exposed to great variations of temperature. Prev. ill. 7 weeks. Pain in hip, and back of right thigh, and calf, increased by pressure (88 days).

Quinine (*gr* i *doses*), *and subdermic injection of morphia, at first Then with* 5 *gr. doses of quinine gradual'y increased to* 15 *grs. t. d. This relieved the pain, but caused slight cinchonism after two days, and was then left off. The pain returning, he was put on quinidine,* 15 *grs. t. d. This did not cause cinchonism and he recovered perfectly under it.*

*Sciatica.—J. W., æt. 52. Carpenter. Adm. Dec. 12, 1862. Disch. Jan. 14. In H. 33 days. *Recovery.* Dr. Johnson, vol. 15, p. 33. Prev. ill. 1 month. Pain along left sciatic nerve, extending down leg. Commenced suddenly. Duration of pain 61 days. Bronchitis on adm.

Subdermic injection of morphia, and blisters to hip. Carbonate of ammonia, and chloric ether at first, and then hydrochlorate of ammonia, and tincture of sesquichloride of iron.

Variola.*—M. P., æt. 38. Hospital-nurse. Adm. Feb. 24. Disch. Mar. 19. In H. 23 days. *Recovery.* Dr. Beale, vol. 6, p. 106. Attended a fatal case of small-pox a fortnight bef. adm. Prev. ill. 6 days, with frontal headache, and pain in stomach and back. Shivering on Feb. 21. Eruption on Feb. 23; slight, not confluent, commencing on right shoulder, then on face and body.

Ammonia and chloric ether. Nitric acid and bark during convalescence.

Chorea.—Ellen W., æt. 8½. Adm. Jan. 5. Disch. Jan. 6, at request of mother. Dr. Garrod, not in case-book. Chorea 3 wks. after a fright, commencing in legs. No heart-disease.

Not treated.

*Chorea and Acute Rheumatism.—L. F., æt. 17. Domestic servant. Adm. Sept. 17, 1862. Disch. Jan. 23. In H. 128 days. *Recovery.* Dr. Budd, vol. 73, p. 30. Catamenia suppressed 2 mon. Prev. ill. 3 wks. A fright, followed by violent choreic movements till 48th day; then slight twitching of hands and face till Jan. 14. Acute rheumatism on 9th day after adm. (37 days) commencing in left wrist, then in ankles and shoulders. Profuse sweating. Systolic bruit over apex of heart.

Quinine, sesquichloride of iron, and shower-baths. During the rheum. a'kalies and bark, morphia, and blisters above joints. After Jan. 1, *sulphate of iron* (8 *grs. t. d. gradually increased to* 16 *grs. t. d.*).

* The patient could not be received at the Small Pox Hospital. See note on page 91.

Mental Derangement.—J. B., æt. 22. Coach-painter. Adm. Dec. 31, 1862. Disch. Jan. 16. In H. 16 days. *Not relieved.* Dr. Johnson, vol. 15, p. 43. Very intemperate. Not violent. Various delusions.
Quinine, and sesquichloride of iron and opium.

Mental Derangement, Epilepsy.—M. W., æt. 33. Groom. Adm. Jan. 19. Disch. Feb. 5. In H. 17 days. *Not relieved.* Dr. Johnson, vol. 16, p. 79. Had syphilis. Prev. ill. 1 yr. Epileptic fits gradually increasing in frequency. Pain in forehead, impaired memory, incoherence, violent at night. No fit while in K. C. H.
Opiates, iodide of potassium and quinine.

Cephalalgia.—E. H., æt. 33. Labourer. Adm. Jan. 27. Disch. Feb. 4. In H. 8 days. *Not relieved.* Dr. Johnson, vol. 16, p. 93. Syphilis 11 yrs. ago. Headache, vertigo, and failure of sight of left eye (2 yrs.). Pupils equal.
Iodide of potassium and morphia.

*Disease of Larynx.—E. C., æt. 40. Employed in a seed warehouse. Adm. Mar. 13. *Died* on Mar. 14. Dr. Johnson, vol. 15, p. 102. Cough during past winter. Prev. ill. 1 wk. with increase of cough and loss of voice. On adm. breathing difficult, not stridulous—no pain over larynx—aphonia. P. above 100. Chest resonant. An attack of spasm a few hours after adm. Tracheotomy. Death shortly after insertion of tube. P.M.—Posterior part of right true vocal cord pushed up by a small hard tumour just below it, size of a large pea, with ulcerated surface, and scirrhous appearance on section. Laryngeal mucous membrane natural. Lungs congested.
Brandy, liq. morph. mur. mxx., steam inhalation, and operation as above.

Mental Derangement.—A. C., æt. 17. Servant. Adm. Jan. 27. Disch. Mar. 28. In H. 60 days. *Improved.* Dr. Johnson, vol. 15, p. 59. Catamenia irregular. Leucorrhœa. Religious delusion. Operated on for strabismus of right eye on Mar. 10.
Opiates, valerian, quinine and sesquichloride of iron, and asafœtida.

Headache.—J. G., æt. 40. Fly Driver. Adm. Jan. 14. Disch. Jan. 28. In H. 14 days. *Relieved.* Dr. Johnson, vol 15, p. 57. Prev. ill. 6 mos., with nausea, loss of appetite, and pain in back of head. Præcordial pain, 4 mos. Hæmatemesis 19 days bef. adm. Muscular tremors, vertigo and sleeplessness.
Opiates and quinine.

*Arachnitis.—T. S, æt. 16. Porter. Adm. Mar. 9. *Died* on Mar. 9. Dr. Johnson, vol. 15, p. 88. Excitable. On Mar. 8 headache and nausea, but able to work a little till afternoon of next day, when the headache increased, he became very restless, and shortly afterwards delirious. On adm. (about 7·30 p.m.) pupils dilated; severe headache; great restlessness; replies "Yes, sir," to almost every question; delirium. Sudden death about ½ an hour after adm., preceded by a slight convulsive attack. P.M.—Pus beneath dura mater, and on surface of convolutions. Kidney congested.

*Delirium Tremens.—M. Q., æt. 39. Betting Man. Adm. Mar. 1. Disch. Mar. 11. In H. 10 days. *Recovery.* Dr. Garrod, vol. 1, p. 60. Sometimes drinks Oij brandy in a day. Drunk 5 days. Prev. ill. 1 day. Nausea; violent tremors; spectral delusions; sleepless. P 120. Noisy on 2nd day after adm. Slept after. Tr. Digit. ℥ij.
A purge; carbonate of ammonia; Liq. Morph. Mur. ℥ij *(in 2 doses); opii gr.* i. *3tiis horis (4 doses). Tr. Digit.* ℥ij, *repeated next night. Porter* ℥i, *brandy* ℥ij.

*Coma (Epileptiform)—Pleurisy.—M. B., æt. 22. Prostitute. Adm. Jan. 8. Disch. Mar. 4. In H. 55 days. *Recovery.* Dr. Garrod, vol. 1, p. 13. Had one child. Occasional headaches (2 yrs.). Catamenia absent (6 wks.). Much mental anxiety of late. Between 2 and 3 p.m. on Jan. 7, while talking to a friend fell down in convulsions, which lasted all night and were followed by stupor. On adm. (2nd day) eyes closed; complete unconsciousness; pupils equal, and sensible to light; slight trismus. No paralysis of limbs. Swallows liquids. Respiration quiet. P. 108. Basic systolic bruit over heart. Urine retained. No albumen. Twenty-three convulsive attacks the next day, between 6 a.m. and 1 p.m. Pupils sensible to light; mucous rattle in throat; motions passed under her; incontinence of urine (11 days). Return of convulsions at 6 p.m.; they ceased at 3 p.m. the next day (4th), and she then, for the first time since admission, noticed what went on around her. On 5th day she answered questions, but was delirious for 5 days following. On 7th day, p. 120; dulness over right base; and on 8th day friction sound over right base (5 days). Violent delirium on 12th day. Rapid improvement after 14th day, though great anœmia remained. Reappearance of catamenia on Feb. 23.

A turpentine enema on adm., and then nothing else, except brandy and beef tea, till 4th day, when 2 drops of croton oil with castor oil, an enema with 15 grs. quinine, and an enema with ʒss of Tr. asafœtida were exhibited. She improved after this, and the asafœtida was repeated on the three following days. Then carbonate of ammonia and bark, and during convalescence quinine and sulphate of iron, and fer reduit. Brandy 6 oz.; increased to 12 oz. on 6th day, and reduced to 6 oz. on 22nd day.

*Paraplegia.—G. W., æt. 29. Soldier. Adm. Dec. 26, 1862. *Died* on Mar. 11. In H. 75 days. Dr. Johnson, vol. 16, p. 59. Primary syphilis 8 yrs. ago; no secondary symptoms. Vertigo at Gibraltar 16 mos. ago: 1 mo. after this, sudden loss of motion and sensation in right arm and leg; face affected; partial recovery. Five months ago, sudden motor palsy of right side; speech slightly affected. Two months ago fell down; no loss of consciousness; legs palsied; sensation and speech unaffected. Tremors of right leg, and slight rigidity of right biceps femoris. Motions and urine passed under him for 26 days after adm. Then rapid improvement, and on 34th day after adm. could walk with a stick. Death sudden, preceded by headache (1 wk.). P. M.—Recent hœmorrhage at base of brain, extending down cord; softening of lower dorsal region of cord. Oily granular cells through whole length of cord.

Quinine and sesquichloride of iron for 5 days; then iodide of potassium, 61 days. During the last wk. quinine and sulphuric acid.

Constipation. Epilepsy.—Clara S., æt. 7. Adm. Jan. 5. Disch. Jan. 18. In H. 13 days. *Relieved.* Dr. Johnson, vol. 16, p. 71. Always epileptic. Fits have diminished in number and severity the last year or two. Drags right leg slightly in walking. Constipation and vomiting.

Castor oil.

*Hemiplegia.—C. R., æt. 43. Butcher's wife. Adm. Jan. 19. Disch. March 11. In H. 51 days. *Slightly Relieved.* Dr. Johnson, vol. 16, p. 81. Scarlet fever when a child. Previous illness 14 days. Mental anxiety. Fell down while at work. Loss of consciousness. Right side paralysed. Complete loss of sensation in right half of body. Mouth and tongue a good deal affected. Slight rigidity of biceps of arm. Basic systolic bruit over heart. Trace of albumen in urine. Gout on 7th day after adm. Slight improvement of leg.

Treatment various, chiefly iodide of potassium, and quinine and iron. Blisters to nape of neck.

*Hemiplegia.—W. F. M., æt. 38. Law clerk. Adm. Dec. 19. Disch. Jan. 17. In H. 29 days. *Relieved.* Dr. Johnson, vol. 16, p. 36. A sun-stroke while in China 10 years ago. Giddiness, dimness of sight, and pains in head, 2 months.

Mental anxiety. During dinner on Dec. 19, sudden loss of consciousness. On adm. unconscious (3 days), loss of motion, and sensation on left side. Not much face palsy. Reflex action unimpaired. No rigidity. Heart healthy. Sensation began to return in arm on 5th day. Recovery nearly complete.

Sal volatile and a purge. Then chloric ether and muriatic acid, and afterwards sesquichloride of iron.

*Paralysis.—T. W., æt. 48. Saddler. Adm. Jan. 21. Disch. Jan. 31. In H. 10 days. *Relieved.* Dr. Beale, vol 5, p. 103. Formerly a soldier. Intemperate. Prev. illness 14 days. Pains in legs. Thickness of speech. Double vision (for 5 days after adm.). Slight loss of power in left arm. Since readmitted with left hemiplegia.

Dilute nitric acid and chloric ether.

*Hemiplegia.—W. L., æt. 23. Sailor. Adm. Feb. 21. Disch., at his own request, Mar. 9. In H. 16 days. *Relieved.* Dr. Johnson, vol. 16, p. 123. Had cholera 9 years ago. Ague 6 years ago. Syphilis 5 years ago. No secondary symptoms. Prev. illness 7 weeks. Four epileptic fits in 2 days, followed by complete loss of motion and partial loss of sensation in left arm and leg On adm. slight motor palsy of face, tongue, arm, and leg. Since readmitted with *right* hemiplegia.

Iodide of potassium.

*Hemiplegia.—H. C., æt. 50, jeweller. Adm. Oct. 2, 1862. Disch. Feb. 21. In H. 142 days. *Relieved.* Dr. Johnson, vol. 13, p. 241. Intemperate. Subject to gout 16 years. Last attack 5 months ago. Pain in back of head 14 days. On morning of adm., after working all night, giddiness, pain in left temple and sudden left hemiplegia, without loss of consciousness. On adm. in complete palsy of motion; reflex action and sensation perfect; slight rigidity of left biceps of arm; headache; right pupil dilated (6 days); urine albuminous; heart normal; radial arteries rigid. From Oct. 4 to Oct. 9, delirium, torpor, and evacuations passed under him. On Oct. 10, an attack of gout commencing in left foot (47 days). Another attack of gout in feet on December 26, and on Jan. 10 in left knee. Recovery nearly complete.

Quinine, and blister to nape of neck, and then ammonio-citrate of iron, chloric ether and colchicum. Blisters to left knee. Brandy 3 *oz.*

*Hemiplegia.—Emma R., æt. 11. Adm. Dec. 14, 1862. Disch. Jan. 31. *Relieved.* In H. 48 days. Dr. Johnson, vol. 15, p. 31. A fright 4 mos. ago, was followed immediately by tremors, and in 3 days by complete loss of power in left arm and leg (9 weeks). On Dec. 14 sudden insensibility and left hemiplegia after a meal of roast pork. Face drawn. Sensation unaffected. Slight rigidity of limbs. Systolic bruit over apex of heart. Partial recovery. A second slighter attack of left hemiplegia, without loss of consciousness, on 16th day after adm. Recovery nearly complete.

Grey powder and rhubarb, and sesquichloride of iron.

*Hemiplegia.—Margaret R., æt. 56. Adm. Jan. 16. Disch. March 18. In H. 62 days. *Slightly relieved.* Dr. Beale, vol. 5, p. 96. Typhus 42 yrs. ago. Right hemiplegia 2 years ago; complete recovery in 1 yr. Three mos. ago tingling and loss of power in left leg (1 wk.). Previous illness 5 wks., sudden loss of power in arm and weakness of leg. On day bef. adm. fell down. Slight loss of consciousness. Complete left hemiplegia, with partial loss of sensation. No rigidity. Reflex action unimpaired. Heart normal.

Treated with quinine and sesquichloride of iron; iodide of potassium; galvanism; and occasional purgatives.

W. K., æt. 50. No occupation. Adm. Mar. 29. Discharged Mar. 30. *Recovery.* Dr. Johnson, not in case book. Slight hemiplegia after drinking.

Neuralgia.—G. U., æt. 63. Printer's Labourer. Adm. Feb. 7. Disch. Feb. 25. In. H. 18 days. *Relieved.* Dr. Johnson, vol. 16, p. 111. Intemperate. Much exposed to cold. Acute rheum. 30 yrs. ago. Neuralgia of face (right side) 3 yrs. Inflammation of face (8 days) following application of a horseradish poultice two days bef. adm.

Carbonate of ammonia, chloric ether and bark; then quinine and sesquichloride of iron. Morphia at night, and local application of belladonna.

Neuralgia.—A. R., æt. 37. Servant. Adm. Mar. 4. Disch. Mar. 25. In H. 21 days. *Recovery.* Dr. Beale, vol. 5, p. 136. Neuralgia of left side of face 3 yrs. In K. C. H. in May, 1862, under Dr. Budd, for same complaint.

Sesquichloride of iron and purgatives.

Paralysis of Third Nerve.—W. F., æt. 31. Plasterer. Adm. Jan. 19. Disch. Jan. 20, at his own request. Dr. Johnson. Not in case-book.

Ulceration of Soft Palate.—Ann C., æt. 36. Servant. Adm. Mar. 3. Disch. Mar. 28. In H. 25 days. *Recovery.* Dr. Johnson, vol. 15, p. 115. Since her marriage, 16 yrs. ago, loss of health, and pains in bones; no eruption. Sore throat 7 yrs., worse during last 6 mos. On adm. uvula and part of soft palate gone. Ulceration extending to hard palate. Sloughing of back of pharynx. Dysphagia.

Iodide of potassium and bark, and local application of nitric acid.

Ulceration of Throat.—S. B., æt. 31. Labourer. Adm. Feb. 28. Disch. Mar. 21. In H. 21 days. *Nearly well.* Dr. Johnson, vol. 15, p. 113. Syphilis 9 yrs. ago, and again 3 yrs. ago. In St. George's 9 mos. ago for secondary eruption. Prev. ill. 3 mos. Ulceration of uvula and right tonsil.

Iodide of potassium, and local application of nitrate of silver, and nitric acid.

Cynanche Tonsillaris.—E. H., æt. 23. Servant. Adm. Feb. 18. Disch. Mar. 11. In H. 21 days. *Relieved.* Dr. Beale, vol. 5, p. 126. Sore throat (14 days) 6 mos. ago. Prev. ill. 9 days. Followed by chronic enlargement.

Chlorate of potass, and sesquichloride of iron. Grey powder and rhubarb twice a week, and sol. A.N. to tonsils.

Cynanche Tonsillaris.—E. S., æt. 17. Servant. Adm. Jan. 22. Disch. Feb. 11. In H. 20 days. *Recovery.* Dr. Beale, vol. 5, p. 108. Prev. ill. 14 days.

Acetate of ammonia and chloric ether, and afterwards dilute nitric acid. Calomel purge. Wine 4 *oz.*

Colic.—M. S., æt. 26. Commissionaire. Adm. Mar. 5. Disch. Mar. 9. *Recovery.* Dr. Johnson, vol. 15, p. 123. Colic after a long walk.

A warm water enema, opium and purgatives.

*Lead Colic.—Ann F., æt. 32. In white lead factory. Adm. Dec. 29, 1862. Disch. Jan. 21. In H. 23 days. *Recovery.* Dr. Johnson, vol. 16, p. 52. Works in a part of the factory where there is much white-lead dust floating about in the air. Loss of appetite for 3 wks. before adm. Pains in limbs, constipation and gradually increasing abdominal uneasiness and pain for 2 wks. before adm. Marked blue line on gums. No dropping of wrists.

Iodide of potassium; colocynth pills; and colocynth and ext. of nux vomica.

Dysenteric Diarrhœa.—W. J., æt. 40. Bricklayer. Adm. Feb. 19. Disch. Mar. 16. In H. 25 days. *Recovery.* Dr. Beale, vol. 5, p. 127. Diarrhœa (3

wks.) 4 mos. ago. Prev. ill. 8 days. Loose motions containing blood; purged every ¼ of an hour at first, and on adm. 3 or 4 times a day. Stools natural on Mar. 6.

Sulphate of copper and opium for 10 days. Then sesquichloride of iron, and castor oil.

*Acute Renal Dropsy.—Caroline C., æt. 36. Adm. Feb. 26. *Died* on Mar. 28. In H. 30 days. Dr. Johnson, vol. 16, p. 127. Dropsy of feet and ankles 10 yrs. ago (4 wks.). Prev. ill. 3 wks. Dropsy of legs. Urine scanty; turbid with lithates; ⅔ths albumen. No thoracic disease. Improved up to 15th day after adm. Then sloughing about left ankle, and back of leg, accompanied by rapid wasting. Death from exhaustion. P. M.—Heart small. Lungs gorged. Liver fatty; wt. 4½ lbs. Kidneys.

Acetate of ammonia and citrate of iron, diuretics, and purgatives. Hot air baths and dry cupping on alternate nights. Right leg punctured on 22nd day after a slough had formed on the left leg.

*Acute Renal Dropsy.—H. A., æt. 27. Carman. Adm. Jan. 31. Disch. Mar. 2. In H. 30 days. *Recovery.* Dr. Garrod, vol. 2, p. 25. Temperate. Much exposed to wet and cold. Prev. ill. 11 days. Pain in belly and right loin (13 days). Vomiting. Scanty, smoky urine; ½ albumen on adm. Dropsy of legs, 24 days.

Cream of tartar and jalap, and mustard poultices to loins. Afterwards sulphate of iron.

*Renal Dropsy.—T. K., æt. 43. Compositor. Adm. Jan. 31. Disch. Mar. 28. In H. 56 days. *Relieved.* Dr. Johnson, vol. 16, p. 95. Syphilis 12 yrs. ago. Prev. ill. 2 mos. Dropsy of feet and ankles spreading upwards to knees. Urine ⅛th albumen, containing blood.

Acetate of ammonia; compound jalap powder; hot air baths; and iron.

*Renal Dropsy.—J. R., æt. 28. Labourer. Adm. Nov. 8, 1862. *Died* on Feb. 26. In H. 110 days. Dr. Johnson, vol. 15, p. 3. Intemperate. Acute rheum. 4 yrs. ago. Prev. ill. 6 wks. Dropsy of face and legs 3 wks. after getting wet. Dyspnœa 1 wk. bef. adm. Anasarca and ascites. Cough and expect. Rhoncus over lungs; dulness over left lower lobe. Heart normal. Urine ½ albumen containing granular and waxy casts, epithelium and blood. A week after adm. 3 epileptic seizures, chiefly affecting left side. Legs punctured on Jan. 8. On Feb. 5 large waxy and granular casts and much oil. Feb. 25, nausea; tongue dry and brown; 10 oz. water in 24 hours. Conscious till a few minutes before death. P. M.—Lungs everywhere adherent. Left ventricle hypertrophied; nitral valve thickened. Much fluid in peritoneum. Commencing atrophy of kidneys.

Dry cupping over loins; hot air baths; hydragogue purgatives, iron, and stimulants. Gin 6 oz.

*Renal Dropsy.—E. J., æt. 29. Tanner. Adm. Dec. 3, 1862. Disch. Jan. 17. In H. 45 days. *Improved.* Dr. Budd, vol. 73, p. 108. Temperate. Previous health good. Prev. ill. 6 wks. Shivering and retching followed by a rash (child with scarlatina in same house). Anasarca; scanty and bloody urine; pain in loins (5 wks.); urine containing oily casts and blood; albumen ½.

Jalapine and colocynth, acetate and carbonate of ammonia, and hot air baths, for a month. Then large doses of jalap and cream of tartar, and sulphate of iron.

Renal Disease.—E. B., æt. 43. Groom. Adm. Mar. 11. Disch. Mar. 28. In H. 17 days. *Relieved.* Dr. Johnson, vol. 16, p. 143. Formerly intemperate. Syphilis 15 years ago. In K.C.H. 6 times under Dr. Budd with same symptoms as at present. Frequent vomiting, 10 wks. Cough and slight hœmoptysis. Tenderness on pressure over abdomen. Granular casts in urine; no albumen.

Acetate of ammonia and chloric ether. Afterwards citrate of iron.

*Renal Dropsy.—Lydia M., æt. 16. Adm. Nov. 26, 1862. *Died* on Jan. 11. In H. 46 days. Dr. Johnson, vol. 15, p. 19. Typhus 6 yrs. ago. Prev. ill. 9 mos. Pain in loins, dropsy of legs, and cough. Heart normal. Urine ⅓rd albumen, containing oily casts, and epithelium. Ascites by 19th day after adm. Erysipelas of right thigh on 29th day (5 days). Pain and tenderness over abdomen on 42nd day, followed by sinking. P. M.—A little lymph on peritoneum; fluid in pleuræ and abdomen; scattered tubercles in lungs; fatty degeneration of kidney.

Jalapine and colocynth, hot air baths, iron and stimulants.

*Renal Disease—Gout.—A. M., æt. 53. Needlewoman. Adm. Jan. 2. Disch. Mar. 4. In H. 61 days. *Recovery.* Dr. Garrod, vol. 1, p. 1. Intemperate. Mania 20 yrs. ago (3 yrs.). Two months ago, pain in loins, debility, vomiting. One month ago, debility and giddiness. Pain, œdema, and redness of feet till 6th day after adm. Urine albuminous (4 days); waxy and granular casts. On Feb. 7, excess of uric acid in blood serum. Scabies.

Treatment, various.

Jaundice.—J. S., æt. 9. Adm. Dec. 29, 1862. Disch. Jan. 14. In H. 16 days. *Recovery.* Dr. Johnson, vol. 15, p. 41. Never had rheumatism. Prev. ill. 8 days. Motions pale. No hepatic pain or enlargement. Systolic bruit at apex of heart. Duration of jaundice 18 days.

Sulphate of magnesia and muriatic acid for a week, and then with ammonio citrate of iron.

Jaundice.—W. K., æt. 19. Printer. Adm. Mar. 5. Disch. Mar. 21. In H. 16 days. *Recovery.* Dr. Beale, vol. 6, p. 113. Prev. ill. 1 wk. Slight jaundice and ulcerated sore throat. Motions pale. No hepatic pain, tenderness, or enlargement.

Nitro-muriatic acid, mist, alba, and quinine.

Jaundice.—A. J., æt. 16½. Housemaid. Adm. Dec. 9, 1862. Disch. Jan. 10. *Recovery.* Dr. Budd, vol. 73, p. 110. Hysterical. Pain in right side and occasional constipation 9 mos. Prev. ill. 3 mos. Vomiting, loss of appetite, and pain and tenderness over right hypochondrium. Jaundice on day after adm. (10 days). Liver enlarged. Constipation.

Sulphate and carbonate of magnesia and sal-volatile. Afterwards quinine and sesquichloride of iron.

Jaundice.—W. L., æt. 16. Printer. Adm. Feb. 4. Disch. Feb. 17. In H. 13 days. *Recovery.* Dr. Johnson, vol. 15, p. 87. Works in an atmosphere loaded with steam. Jaundice 1 wk. bef. adm., preceded by nausea and vomiting. Headache. No pain, tenderness, or enlargement of liver. Motions clay coloured. Duration of jaundice, 19 days.

Pil. panis for 1 wk. Then mist. alba and quinine and sulphuric acid.

Jaundice.—H. M'Q., æt. 27. Stationer. Adm. Feb. 23. Disch. Mar. 14. In H. 19 days. *Recovery.* Dr. Johnson, vol. 15, page 109. Acute rheumatism 4 yrs. ago. Nausea and vomiting on Feb. 11. Constipation. Jaundice 1 wk. before adm. Languor. Motions clay coloured. No hepatic pain, tenderness, or enlargement. Systolic bruit over apex of heart.

Sulphate of magnesia; dilute nitro-muriatic acid; and podophyllin and colocynth.

Jaundice.—A. S., æt. 16. Lithographic Printer. Adm. Jan. 20. Disch. Feb. 11. In H. 12 days. *Recovery.* Dr. Garrod, vol. 2, p. 22. Sour risings 1½ yrs. Costive bowels, 3 wks. Prev. ill. 6 days. "Caught cold;" feeling of constriction round belly, and jaundice. Slight hepatic tenderness on day after adm.; no enlargement. Motions pale till 4th day after adm.

Calomel purge, and sulphate of magnesia, and sal-volatile.

*Cirrhosis of Liver.—Eliz. F., æt. 51. Cook. Adm. Feb. 18. *Died,*

Mar. 14. In H. 24 days. Dr. Johnson, vol. 16, p. 119. Been a spirit drinker. No rheum. Prev. ill. 7 wks. Gradual painless enlargement of abdomen. Skin dry; superficial abdominal veins enlarged; considerable ascites, liver pushed up. Systolic bruit over apex of heart; urine loaded with lithates; not albuminous. Two gallons of fluid drawn off by tapping on Mar. 6. Seven days after, peritonitis, and rapid sinking. P. M.—About Oiv fluid in abdomen; recent lymph on intestines. Liver cirrhose, wt. 3 lbs. 2 oz. Large firm spleen, wt. 18 oz. Kidneys granular. Heart healthy.

Diuretics and stimulants, and operation as above.

*Hepatic Disease.—T. H., æt. 37. Shoemaker. Adm. Jan. 21. Disch., at own request, Mar. 12. In H. 50 days. *Relieved.* Dr. Beale, vol. 5, p. 104. Had syphilis 18 yrs. ago, followed by secondary symptoms. Sudden stabbing pain in right side 1 yr. ago. Six months ago an attack resembling dysentery, followed, after 14 days, by pain and swelling in region of liver, and pain in right shoulder. About two mos. ago a second dysenteric attack. Admitted under Dr. Budd on Dec. 11, and remained in hosp. 19 days. Sallow and wasted. Coppery eruption on skin. Constant pain in right side. Liver extending to 2 in. below umbilicus, and nodulated on surface. Albuminous urine. Constant salivation. Nausea after food. Waterbrash. Constipation.

Hydrochlorate of ammonia for 4 days, and then hydrocyanic acid and bicarbonate of soda. Mercurial ointment rubbed in over liver.

*Gout.—G. P., æt, 31, actor. Adm. Mar. 9. Disch. Mar. 25. In H. 16 days. *Recovery.* Dr. Garrod, vol. 2, p. 70. A free liver. Had two slight attacks of delirium tremens. No hereditary predisposition to gout. First attack in right great toe 9 years ago. Slighter attacks since. Severe general gout a year ago, and every 6 weeks since. Previous illness 9 days. Commenced in right hand, wrist, and elbow; then right foot, left hand, and knees. Slight excess of uric acid in blood serum. Chalk stone on 2nd joint of right fourth finger. Urine not albuminous. No thoracic disease. No pain after 21st day.

Bicarbonate of potass, colchicum and sulphate of magnesia. Afterwards ammoniated tincture of guiacum and iodide of potassium.

*Purpura.—H. C., æt. 21. Light porter. Adm. Jan. 20. *Died* on Feb. 2. In H. 13 days. Dr. Garrod, vol. 2, p. 9. No deficiency of vegetables. Father gouty. Loss of flesh 3 mos. Epistaxis on and off 6 weeks. Purpuric spots on legs 4 weeks. Headache. Gums spongy and bleeding; blood in motions and urine. Systolic bruit over base of heart. Tongue dry and brown. P. 136. Delirium on 12th day after adm. Tenderness on pressure over abdomen, and motions and urine passed under him on 13th day. P. M.—Recent lymph on liver. Pus in peritoneal cavity. Purpuric spots on all the viscera. Spleen friable, wt. 11b. 13 oz. Extensive old disease of aortic valves.

Camphor mixture for 2 days. Then with large doses of sulphate of iron, and for the last 2 days gallic acid. During 2nd week wine 6 oz.; changed for brandy 8 oz. on 11th day.

*Purpura.—M. B., æt. 67. Charwoman. Adm. Jan. 17. *Died* on Jan. 25. In H. 8 days. Dr. Garrod, vol. 2, p. 6. For last 3 mos. lost appetite, and lived chiefly on biscuits and porter. Prev. illness 8 days. General purpuric eruption, commencing on arms, where large patches formed. No soreness or bleeding of gums. Dark motions. Tongue dry and brown. Pulse 100 feeble and irregular. Loud rubbing sound over heart on 2nd day after adm. (7 days). No increase of cardiac dulness. On 5th day after adm. dulness and crepitus over left base. Delirium for 2 days before death. P. M.—Wt. of heart 15 oz; recent lymph on its surface; Left vent. hypertrophied. Lower lobes of lungs firm and sank in water.

Beef tea diet, slice and vegetables, wine 4 oz. and mist. camph. for three days.

Then quinine and opium and wine 8 *oz. Cardiac region painted with iodine. On 7th day sal-volatile and ammonia and brandy* 12 *oz.*

PURPURA.—C. E., æt. 21. Kitchenmaid. Adm. Jan. 19. Disch. Jan. 31. In H. 12 days. *Recovery.* Dr. Beale, vol. 6, p. 76. Previous general health good. No deficiency of vegetable food before adm. Prev. illness 1 week. Purpuric eruption on legs and feet, accompanied by pain and stiffness. No sponginess of gums.

Sesquichloride of iron.

*CHRONIC ECZEMA.—Catherine G., æt. 3. Adm. Feb. 4. Disch. Feb. 28. In H. 24 days. *Relieved.* Dr. Beale, not in case book. Eczema of face and arms. Admitted under Dr. Beale on Sept. 25, 1862, with eczema of face, scalp, neck, arms, and legs; remained in H. 97 days, and was nearly well on discharge.

Glycerine and bicarbonate of potass, grey powder and rhubarb, and local application of nitrate of bismuth ointment.

*CHRONIC ECZEMA.—H. B., æt. 14. Bricklayer's boy. Adm. Jan. 20. Disch. Mar. 14. In H. 53 days. *Relieved.* Dr. Johnson, vol. 16, p. 83. Eczematous eruption on face and scalp 12 years on and off. Abscess below right ear. General health never very good.

Cod liver oil and liq. potass arsenitis (m iij *gradually increased to m* vij). *Locally oxide of zinc and glycerine.*

*ECZEMA.—S. C., æt. 20. Kitchenmaid. Adm. Jan. 2. Disch. Mar. 21. In H. 78 days. *Recovery.* Dr. Johnson, vol. 15, p. 45. Prev. illness 3 weeks. An abscess in right forearm, followed by eczema, spreading to face and left arm.

Treated with sesquichloride of iron for nearly a month, and then Fowler's solution. Grey powder and rhubarb. Locally glycerine lotion and bismuth ointment.

*ECZEMA.—Wm. M., æt. 12. Adm. Dec. 5, 1862. Disch. Jan. 24. In H. 52 days. *Recovery.* Dr. Johnson, vol. 15, p. 27. Prev. illness 8 mos. Eczema of skin over right popliteal space, forming an oval patch about 12 inches long. Small abscess on inside of thigh.

Sesquichloride of iron. Locally Ung. Bismuth. Nitrat. at first, and afterwards equal parts white precip. ointment and compound lead cerate.

ECZEMA.—M. B., æt. 25. Married. Adm. Mar. 30. Dis. Mar. 31. *Made O. P.* Dr. Beale, not in case book. Eczematous eruption on hands and face (3 months).

Not treated while in hospital.

*J. B., æt. 49. Butcher. Adm. Jan. 28. Disch. Mar. 25. In H. 56 days. *Relieved.* Dr. Garrod, vol. 1, p. 30. Gin drinker. Prev. ill. 1 yr. Frequent loose, pale, slimy stools; containing fat (till Mar. 21) and sometimes blood. Pain after food: waterbrash; occasional vomiting; loss of flesh (6 mos.). Headache (2 mos.). Œdema of legs.

Oxide of silver. Afterwards citrate of iron, and cod liver oil.

*G. B., æt. 41. Silk Weaver. Adm. Jan. 7. Disch. Jan. 22. In H. 15 days. *Relieved.* Dr. Garrod, vol. 1, p. 11. No history of ague. Prev. ill. 4 months. Sudden sharp pain over spleen (4 wks.) followed by a swelling in left hypochondriac and part of left lumbar, umbilical and epigastric regions. Through this the heart's sounds heard distinctly. Six lowest left intercostal spaces filled up. Dulness or percussion over this space, and thickening of skin. His occupation does not cause any undue pressure on this part.

Syrup of iodide of iron, and iodine paint over swelling.

POISONING BY OXALIC ACID.—Mary M'M., æt. 63. Adm. Mar. 9. Disch. Mar. 14. *Recovery.* Dr. Beale. Not in case book. Cup taken away from her before much had been swallowed. Lips and tongue swollen and inflamed.

T. R., æt. 33. Labourer. Adm. Dec. 31, 1862. Disch. Jan. 7. *Recovery.* Dr. Johnson. Not in case book. Half starved. Prev. ill. 14 days. Swelling and redness of legs, with great general depression.
Carbonate of ammonia, chloric ether and bark; wine and nourishing diet.

Debility.—C. S., æt. 38. Needlewoman. Adm. Jan. 13. Disch. Jan. 28. In H. 15 days. *Relieved.* Dr. Johnson, vol. 16, p. 74. Hard worked and ill fed. Always epileptic: fits very frequent of late. Ten yrs. ago epileptic left hemiplegia. Prev. ill. 14 days. Headache and debility.
Quinine and sesquichloride of iron, and morphia at bed time.

Debility.—Maria P., æt. 60. Adm. Jan. 19. Disch. Jan. 31. In H. 12 days. *Relieved.* Dr. Johnson, vol. 15, p. 67. Winter cough 10 yrs. Prev. ill. 4 yrs. Increasing debility and loss of muscular power. No pain or impaired sensation.
Quinine and sesquichloride of iron.

Hysteria.—Catherine B., æt. 27. Servant. Adm. Feb. 17. Disch. Feb. 25. In H. 8 days. *Relieved.* Dr. Johnson, vol. 15, p. 9. Cough and occasional loss of voice and globus hystericus. Catamenia regular. Tenderness over larynx 1 wk. Interior of larynx normal.
Sesquichloride of iron.

Hysteria.—I. H., æt. 16. Servant. Adm. Jan. 8. Disch. Jan. 21. In H. 13 days. *Relieved.* Dr. Johnson, vol. 16, p. 67. Hard worked. Prev. ill. 2 mos. Debility; suppression of catamenia; pain and tenderness in right hypochondrium where there is an indistinct tumour, disappearing under chloroform.
Iron and gentian, and opiate liniment.

Hysteria.—A. B., æt. 24. Servant. Adm. Feb. 26. Disch. Mar. 25. In H. 27 days. *Relieved.* Dr. Garrod, vol. 1, p. 58. Fits accompanied by globus hystericus. Pregnant 4 mos.
Treated with ammonia and chloric ether, and asafœtida.

Hæteria.—Susan H., æt. 26. Servant. Adm. Dec. 30, 1862. Disch. Jan. 3. In hos. 4 days. Dr. Beale, vol, 5, p. 54. *Recovery.* Hysterical dyspnœa.
Treated with sal volatile and valerian.

Croup.—Emma R., æt. 3. Adm. Feb. 1. *Died* on Feb. 2. Brought in at night. Not in case book. Tracheotomy performed.

*Hæmatemesis.—M. F., æt. 38. Servant. Adm. Nov. 20, 1862. Disch. Jan. 17. In H. 58 days. *Recovery.* Dr. Budd, vol. 74, p. 106. Intemperate. Catamenia irregular. Vomiting, pain after food, and sour risings 5½ years ago, and twice since. Prev. illness 6 mos. Slight hæmatemesis. Pain after food in epigast. and back. Vomiting. Free from pain and vomiting on Jan. 10.
Treated at first with nitrate of bismuth and paregoric. Afterwards with oxide of silver for 3 days. Light diet.

Hæmatemesis.—J. U., æt. 35. "Tight maker." Adm. Feb. 5. Disch. Feb. 20. In H. 15 days. *Recovery.* Dr. Johnson, vol. 16, p. 99. Hæmatemesis (1 qt.) after lifting a heavy weight 2 yrs. ago. Flatulence and sour risings 2 yrs. ago. Prev. illness 3 days. Vomited about 4 oz. of blood. Pain in epigastrium and back. Retching after food. Appetite bad.
Treated at first with turpentine. Then with sesquichloride of iron and chloric ether. Light diet.

Hæmatemesis.—T. H., æt. 31. Labourer. Adm. Jan. 21. Disch. Feb. 4. In H. 14 days. *Recovery.* Dr. Johnsou, vol. 15, p. 73. Small pox 11 years ago. Prev. illness 7 wks. Shivering; pain in right side; cough; vomiting till 10th day after adm. Hæmatemesis and dark motions 6 times in last 5 weeks.

Largest quantity of blood brought up = 0i. Flatulence and constipation. Heart, lungs, and urine normal.

Treated with hydrocyanic acid and bicarbonate of potass and light diet.

Dyspepsia.—A. C., æt. 31. Laundress. Adm. Jan. 20. Disch. Mar. 4. In H. 43 days. *Relieved.* Dr. Johnson, vol. 15. p. 59. Pain across abdomen. Appetite bad. Constipation. Headache.

Treated with purgatives; opium; asafœtida; and then with strychnia.

Dyspepsia.—A. M., æt. 30. Housemaid. Adm. Feb. 18. Disch. Feb. 28. In H. 10 days. *Relieved.* Dr. Johnson, vol. 16, p. 103. Prev. ill. 2 mos. Suppression of catamenia. Pain after food, nausea, vomiting, waterbrash.

Ammonio citrate of iron, soda bicarb., and hydrocyanic acid. Purgatives.

Dyspepsia.—L. L., æt. 30. Needlewoman. Adm. Mar. 3. Disch. Mar. 18. In H. 15 days. *Recovery.* Dr. Beale, vol. 6, p. 111. Pain in epigastrium, and right side. Vomiting. Slight leucorrhœa.

Hydrocyanic acid and bicarbonate of soda, and purgatives.

Dyspepsia.—P. B., æt. 45. Baker. Adm. Feb. 11. Disch. Feb. 25. In H. 14 days. *Recovery.* Dr. Johnson, vol. 15, p. 89. Temperate. No family hist. of cancer. Prev. ill. 14 days. Pain and soreness of epigastrium; vomiting soon after food; waterbrash; constipation.

Bismuth and magnesia, light diet and purgative enema. Afterwards with hydrocyanic acid.

Dyspepsia.—T. H., æt. 39. Railway Guard. Adm. Mar. 16. Disch. Mar. 28. In H. 12 days. *Relieved.* Dr. Johnson, vol. 15, p. 135. Intemperate. Prev. ill. 9 mos. Sour risings after food. Flatulence and pain in epigastrium.

Strychnia and sesquichloride of iron.

Periostitis.—T. F., æt. 38. Labourer. Adm. Dec. 27, 1862. Disch. Jan. 2. *Relieved.* Dr. Beale, vol. 6, p. 57. Secondary syphilitic periostitis, and eruption on scrotum.

Iodide of potassium. Locally, white precipitate ointment.

Exhaustion.—W. J., æt. 32. Labourer. Adm. Feb. 2. Disch. Feb. 7. *Recovery.* Dr. Johnson. Not in case book. Exhaustion from travelling on foot.

Anæmia.—S. B., æt. 22. Nursemaid. Adm. Dec. 20, 1862. Disch. Jan. 28. In H. 39 days. *Recovery.* Dr. Budd, vol. 74, p. 135. Very hysterical. Catamenia regular. Prev. ill. 3 wks. Slight anæmia. Basic systolic bruit over heart, and venous murmur in neck (21 days). Severe pain in right side on Jan. 1 (22 days).

Valerian, sulphate of iron, and asafœtida.

Anæmia.—M. A. L., æt. 20. Servant. Adm. Dec. 3, 1862. Disch. Feb. 3. In H. 62 days. *Recovery.* Dr. Budd, vol. 74, p. 125. Prev. ill. 2½ months. Catamenia irregular. Systolic bruit over base of heart and venous murmur in neck.

Sesquichloride of iron 12 *days, sulphate of iron, and quinine,* 16 *days, and fer reduit,* 34 *days. Aloetic aperients.*

Anæmia.—M. L., æt. 22. Cook. Adm. Jan. 3. Disch. Jan. 21. In H. 18 days. *Recovery.* Dr. Johnson, vol. 15, p. 51. Shortness of breath on exertion, and palpitation, 3 yrs. Scanty catamenia and occasional leucorrhœa, 5 mos. Headache 5 wks. Occasional vomiting 3 wks. Considerable anæmia. Basic systolic bruit, and venous murmur in neck.

Sesquichloride of iron, and sulphate of iron, and ext. gentian.

*Scarlet Fever.—Eliza G., æt. 24. Servant. Adm. Mar. 12. Disch. April 8. In H. 27 days. *Recovery.* Dr. Beale, vol. 5, p. 140. Prev. ill. 4 days. Commenced with shivering, sore throat, and great difficulty of swallowing. Eruption fully out on adm. Cervical glands swollen and painful. Left tonsil and left side of uvula sloughing. Desquamation about 10th day. Severe case.

Liq. ammon. acetatis, and chloric ether, 21 days. Then quinine and sesquichloride of iron. Throat swabbed with glycerine and sesquichloride of iron. Wine 4oz.

Scarlet Fever.—J. C., æt. 23. Married. Adm. April 13. Disch. Ap. 27. In H. 14 days. *Recovery.* Dr. Johnson, vol. 16, p. 169. Suckling 12 months. Prev. ill. 6 days. No evidence of contagion. Commenced with sore throat and headache. Eruption on 2nd day. P. 96. Desquamation on 9th day. Was discharged at her own request before desquamation had ceased.

Liq. ammon. acetatis (7 days), sesquichloride of iron. Throat swabbed with equal parts of glycerine and tinct. sesquichloride of iron.

Scarlet Fever.—Eliza T., æt. 16. Servant. Adm. May 14. Disch. June 13. In H. 30 days. *Recovery.* Dr. Johnson, vol. 16, p. 209. Had measles and small-pox. No evidence of contagion. Prev. ill. 2 days. Commenced with vomiting. Eruption on 3rd day. Slight sore throat. P. 140. Desquamation on 7th day. On 9th day wrists red, swelled, tender, and painful, 2 days. Heart unaffected.

Liq. ammon. acetatis and chloric ether (4 days), sesquichloride of iron (2 days), bicarbonate of potash and quinine and opium (5 days). Hot air baths.

*Scarlet Fever.—Dropsy.—Rosina M., æt. 6. Adm. April 13. Died on May. 10. In H. 27 days. Dr. Beale, vol. 5, p. 154. Scarlet fever in same house. Prev. ill. 5 days. Rash fully out on adm. Desquamation about 14th day. On 17th day very fretful, no appetite, constant vomiting, œdema of face and extremities, and hæmaturia. Epistaxis on 27th and 28th days. Pain and tenderness of abdomen on 30th day; no remission of other symptoms. Death on 32nd day, apparently from exhaustion. No P.M.

Chlorate of potash and dilute muriatic acid (10 days), then sp. ammon. arom., liq. ammon. acetatis, chloric ether, and hydrocyanic acid. Purgatives. Brandy. Dry cupping and sinapisms to loins.

*Scarlatina Puerperalis.—S. C., æt. 25. Married. Adm. May 14. Disch. June 14. In H. 31 days. *Recovery.* Dr. Garrod, vol. 1, p. 108. No previous attack. No evidence of contagion. Confined on May 14th. Restlessness and vomiting on 16th. Sore throat on 18th (1st day). Eruption on 19th. On 3rd day P. 132, R. 44. On 4th day P. 120, R. 60. Tongue red and dry. Rash fading. Delirious since 2nd night. Lochia and milk suppressed. Abscesses over hips and sacrum. Urine slightly albuminous. On 5th day P. 120, R. 40; less delirium, tongue moister, urine still albuminous. On 6th day, rational; no albumen; desquamation commencing. P. 92. On 9th day lochia natural. Quite well on the 25th day. Child not attacked.

Liq. ammon. acetatis and chloric ether. Brandy up to 12 oz. Throat swabbed with glycerine and tincture of sesquichloride of iron.

*Scarlatina Puerperalis.—E. B., æt. 19. Married. Adm. Mar. 18. Disch. April 11. In H. 28 days. *Recovery.* Dr. Garrod, vol. 1, p. 73. No evidence of contagion. Had scarlatina when a child. Headache on Mar. 14 (1st day). Confined the next day. Sore throat in evening. On 5th day, P. 140, eruption fully out, slight sore throat, milk partially suppressed, lochia healthy. On 8th day, wrists and left knee swollen and painful (4 days), desquamation commencing. Child free from eruption. Died in convulsions soon after birth.

Sulphate of magnesia, liq. ammon. acetatis and decoction of bark, 8 days; liq. cinchonæ and chloric ether, 8 days; and then liq. ammon. acetatis and chloric ether. Brandy 6 oz., increased to 8 oz. on 12th day.

*Erysipelas.—James P., æt. 14 days. Adm. May 13. *Died* on May 17. In H. 4 days. Dr. Garrod, vol. 2, p. 121. No evidence of contagion. Mother healthy. Prev. ill. 2 days. Commenced behind right ear with redness and swelling, which, by the next day, had extended over the back of the neck; and on the 3rd day covered the upper part of the back, forming a patch about the size of the palm of an adult hand, swelled and hard, and of a deep red colour. No vesications and no fluctuation anywhere. Motions rather loose By the 5th day the inflammation had spread over the sternum and between the scapulæ, and by the 6th day covered about one-third of the surface of the body. The child took the breast well throughout. Attacked with severe dyspnœa on the 7th day, and died a few hours afterwards. Death preceded by slight convulsions. P.M.—The portion of the skin that had been inflamed was of a purplish tint. Venous congestion of brain and sinuses. Lungs congested. Other viscera healthy.

Ol. Ricini ʒi. *Collodion and castor oil applied to eruption. Mother placed on full diet, with extra pint of porter.*

*Erysipelas.—J. P., æt. 34, diver. Adm. Ap. 30. Disch. May 9. In H. 9 days. *Recovery.* Dr. Johnson, vol. 17, p. 1. Acute bronchitis 3 months ago. Ailing for 14 days before admission. Prev. ill. 2 days. Erysipelas of face and upper part of neck, commencing under left eye. No albumen in urine. Convalescent about 10th day.

Quinine and sesquichloride of iron mixture, purgatives, and collodion and castor oil locally.

*Erysipelas.—J. F., æt. 23, shoemaker. Adm. May 6. Disch. May 23. In H. 17 days. *Recovery.* Dr. Johnson, vol. 17, p. 15. A similar attack 2 yrs. ago. Prev. ill. 4 days. Shivering and vomiting, followed by erysipelatous inflammation of face and scalp, commencing on right cheek. Vesications on ears. P. 112, R. 32. Slight bronchitis. Tongue thickly furred. Convalescent on 17th day. Severe case.

Quinine and sesquichloride of iron. Purgatives. Collodion and castor oil locally. Brandy 6 *oz.*

*Erysipelas.—J. F., æt. 43, omnibus time-keeper. Adm. May 30. Disch. June 17. In H. 18 days. *Recovery.* Dr. Johnson, vol. 15, p. 211. Intemperate. Acute rheumatism 7 yrs. ago. Prev. ill. 5 days. Commenced with pain in right great toe, extending up leg. Two epileptic fits soon afterwards. Then right leg from knee to ancle became red, painful and swollen. No gout. On adm. he was slightly delirious. Tremors, spectral illusions, feeble pulse. Two patches of vesication had formed, one on the front, and the other on the inner side of leg. Urine ¼th albumen, containing granular and oily casts, and scattered pus. No abnormal heart sounds. Slept well on night of admission. On 11th day, still tremulous. On 14th day, albumen increased, and slight œdema of legs.

Opium. Brandy 6 *oz. Chloric ether and carbonate of ammonia. Sesquichloride of iron and quinine locally; fomentations; and collodion and castor oil.*

Variola.*—Anne W., æt. 37. Adm. May 18. Disch. May 30. In H. 12 days. *Recovery.* Dr. Johnson, vol. 17, p. 37. Prev. ill. 3 days. Slight attack. Convalescent on 13th day.

Quinine and chloric ether. Hot-air bath. Morphia.

Variola.*—Mary Anne J., æt. 15. Adm. June 1. Disch. June 3. In H. 2 days. Dr. Beale.

Variola.*—Eliza A. J., æt. 22. Adm. April 18. Disch. Ap. 19. Dr. Beale.

* These cases of variola were treated in the hospital, because the Small Pox Hospital was too full to receive them.

Febricula.—Mary Ann S., æt. 25, dressmaker. Adm. Mar. 23. Disch. Ap. 4. In H. 12 days. *Recovery.* Dr. Beale.

Liquor ammoniæ, acetatis, and tincture of squills. Afterwards quinine and sesquichloride of iron.

Febricula —Daniel F., æt. 10. Adm. Ap. 2. Disch. Ap. 14. In H. 12 days. *Recovery.* Dr. Johnson, vol. 15, p. 153.

Liquor ammoniæ, acetatis, and camphor mixture.

Febricula —Sophia O., works a sewing-machine. Adm. Ap. 8. Disch. Ap. 22. In H. 14 days *Recovery.* Dr. Beale.

Nitro-muriatic acid, liquor cinchonæ, and chloric ether.

Febricula.—Jane S., æt. 17, servant. Adm. May 5. Disch. May 20. In H. 15 days. *Recovery.* Dr. Johnson, vol. 17, p. 11.

Carbonate of ammonia and chloric ether (10 *days*). *Afterwards quinine and sesquichloride of iron.*

Febricula.—Mary B., æt. 25. fruit-seller. Adm. June 15. Disch. June 24. In H. 9 days. *Recovery.* Dr. Garrod, vol. 1, p. 134.

Calomel and colocynth purge. Camphor mixture.

*Typhus.—R. J., æt. 22, shoemaker. Adm. Mar. 18. Disch. Ap. 15. In H. 28 days. *Recovery.* Dr. Garrod, vol. 1, p. 71. Small-pox 1 mo. ago. Convalescent about 12 days before adm., but still very weak. Access of typhus gradual. No evidence of contagion. Prev. ill. about 12 days. On adm., face flushed; considerable tremor of limbs; very deaf; thick rubeoloid rash on abdomen and extremities. P. 140, R. 28. Tongue dry and brown. Delirious 2 days. No diarrhœa. No albumen in urine. Convalescent about the 15th day after adm.

Nitro-muriatic acid liquor cinchonæ, and chloric ether. Afterwards ammonio citrate of iron, and infusion of quassia. Brandy 8 *oz. Increased to* 12 *oz. on 5th day after adm.*

*Typhus.—J. G., æt. 52, lawyer's clerk. Adm. Ap. 15. Died on Ap. 22. In H. 7 days. Dr. Garrod, vol. 1, p. 87. Intemperate. Worked harder than usual during last mo. No evidence of contagion. Access gradual. He gave up work about 6 days before adm. On adm., distinct measly rash on trunk and limbs; tongue coated and dry. P. 112, R. 28. On 5th day after adm., great prostration; delirium; limbs and tongue very tremulous; P. 126, very feeble; R. 36; granular casts, and albumen in urine; an epileptic fit about 1.30 P.M. On 6th day after adm., breathing stertorous. P. 126, R. 36; comatose. Died on 7th day after adm.

Dilute nitro-muriatic acid, liquor cinchonæ, and chloric ether. Brandy 6 *oz. on adm. Increased to* 18 *oz. on 19th, and* 24 *oz. on 20th. Tr. opii* mxv *on 20th. Tr. belladonnæ* mxv 4 *tis horis, on 21st.*

*Typhus.—Emma T., æt. 21, married. Adm. Ap. 23. Disch. May 23. In H. 30 days. *Recovery.* Dr. Johnson, vol. 16, p. 183. Suckling for last 12 mos. No evidence of contagion. Access gradual. Prev. ill. about 14 days. Headache; vomiting; pains in back and limbs; and 5 or 6 loose motions a day. Eruption visible, and delirium on evening of 22nd. On adm., copious rubeoloid eruption on trunk and limbs; tongue brownish yellow; P. 124, R. 36; purged 3 times in 24 hours. On Ap. 24, P. 136, R. 40. Delirious on 25th, 26th, and 27th. Convalescent about May 17.

Dilute muriatic acid, quinine, and chloric ether. Then quinine and dilute muriatic acid. Brandy 6 *oz. Increased to* 16 *oz. on April* 27, *and diminished to* 12 *oz. on May* 1.

*Typhus.—T. H., æt. 49, tailor. Adm. May 23. Died on June 5. In H. 13 days. Dr. Garrod. vol. 2, p. 138. Temperate. Subject to gout. First attack 14 years ago. No evidence of contagion. Prev. ill. 11 days. Violent shivering,

loss of appetite, sleeplessness, and constipation. On adm., thick measly rash on trunk and limbs; tongue dry and brown; P. 124, very feeble; slight headache. Delirious for the first time on night before adm. Urine slightly albuminous. On 19th day, P. 88, R. 28. Semi-conscious; evacuations passed under him. Tongue dry and brown; limbs and tongue very tremulous. Was nearly suffocated by some orange peel, on 21st day. During its removal he struggled a good deal, using the limbs of both sides equally well. The coma became more profound, and he died on 24th day. P.M.—Red softening of right corpus striatum. A cyst as large as a small apple in left kidney. Surface of kidney somewhat granular. Gall bladder contracted on 8 or 10 small stones; other viscera healthy.

Quinine, sulphuric acid, and decoction of bark for 9 days. Then carbonate of ammonia and tincture and decoction of bark. Brandy, 12 oz.

*Subacute Rheumatism. - Typhus.—Trismus.—W. C., æt. 46. Labourer. Adm. Jan. 28. *Died* on Ap. 12. In H. 74 days. Dr. Beale, vol. v., p. 110.—Temperate. Has lived badly of late. Admitted with subacute rheumatism. Prev. ill, 21 days. Commenced in ankles and knees, then in wrists, elbows, and shoulders. Profuse sweating. No cardiac disease. Free from pain on Feb. 17, the left shoulder remaining somewhat stiff. On Mar. 15 (first day of fever), headache, lassitude, loss of appetite, and pains in limbs. There was a case of typhus in the ward about this time. On 3rd day, thick rubeoloid eruption on trunk and extremities. On 6th day P. 120, R. 24. Convalescent from fever on 15th day. On 16th day the right leg and thigh become painful and œdematous; tongue coated. P. 92. Purpuric eruption on legs about 24th day. On 26th day could not separate jaws more than ¼ an inch; masseter and temporal muscles stiff and painful. Right leg as before. On 28th day, trismus much increased; can swallow well. On 29th day, an epileptic fit about 5 A.M.; 24 fits in next 12 hours; rational in the intervals. Died during the last fit. P.M.—Calvarium very thick. Slight opacity of arachnoid. Brain healthy. Firm coagula in right external iliac and femoral veins. Cord not examined.

Acetate of ammonia, bicarbonate of potass, and chloric ether, and quinine and opium. On Mar. 19 liquor cinchonæ, chloric ether, and dilute nitro-muriatic acid. Brandy up to 12 oz. During the trismus, belladonna and Indian hemp and injections of quinine and beef tea.

*Typhoid Fever.—J. W., æt. 27, Telegraph Clerk. Adm. April 27. Disch. May 30. In H. 33 days. *Recovery.*—Dr. Beale, vol. v., p. 151. No evidence of contagion. No marked shivering. Prev. ill about 14 days. Slight diarrhœa before admission. On adm. P. 92, R. 36. Five or six rose spots on chest and abdomen. Tympanitis (till May 18). Slight cœcal tenderness from May 1 till May 18. Purged about three times every day till May 11. Cough, slight mucopurulent expectoration, crepitation over bases of lungs. Convalescent about May 19th.

Liq. cinchonæ, chloric ether, dilute muriatic acid, sp. ammon, co. and decoction of logwood (21 days). Turpentine stupes. Brandy 6 oz. on admission, increased to 12 oz. on April 29, and 16 oz. on May 2. Quinine and sesquichloride of iron during convalescence.

*Typhoid Fever and Pneumonia.—C P., æt. 29. Hospital Nurse. Adm. April 5. Disch. May 11. In H. 34 days. *Recovery.*—Dr. Garrod, vol. i., p. 84. Had "brain fever" seven years ago. Had scarlatina. Some cases of *typhus* in her ward but no *typhoid*. Prev. ill 3 days. Commenced with shivering. On second day vomiting and purging, pain in head, dry cough, and delirium (3 days). Dullness (16 days) and fine crepitation at right base, extending up to angle of scapula on 4th day. On 5th day, P. 112, R. 28. Vomiting on 6th and 7th days. On 10th day some rose spots on back, tongue dry and brown, slight cœcal tenderness (7 days). Abundant rose eruption over abdomen and chest on 12th day.

Purged about 3 times in 24 hours; no blood. Convalescent on 25th day of illness.

Acetate and carbonate of ammonia and chloric ether for a week. Then potash in effervescence. Wine, 4 oz., on adm. changed for brandy, 6 oz. on 15th day (16 days).

*Pneumonia.—H. W., æt. 48. Auctioneer's Porter. Adm. April 3. Disch. May 2. In H. 30 days. *Recovery.* Dr. Johnson, vol. 16, p. 161. Prev. ill. 7 days. Caught cold. Great prostration. Delirium for 4 days after adm. On adm. P. 136., R. 36. Dullness on percussion over left lung behind, from midway between spine and angle of right scapula downwards, with bronchial breathing, bronchophony and diminished vocal vibration. Expectoration viscid, tinged with blood till 18th day. Breathing then bronchial, crepitus heard on a deep inspiration. On 27th day breathing still bronchial about angle of scapula.

Liq. ammoniæ acetatis, and chloric ether (10 days), then quinine and sulphuric acid. Turpentine stupes. Brandy, 6 oz.

*Pneumonia.—W. B., æt. 26. Labourer. Adm. Mar. 4. Disch. April 4. In H. 31 days. *Recovery.* Dr. Beale, vol. v., p. 132. Much exposed to wet and cold. Prev. ill. 4 days. Shivering, pain between shoulders and in right side, cough, and dyspnœa. On adm. dullness on percussion, and bronchial breathing over back of left lung from spine of scapula downwards. Expectoration tinged with blood. On 9th day P. 104, R. 40. Countenance dusky, tongue dry, is much purged. On 33rd day base of left lung still slightly dull; fine crepitus at end of inspiration. No expectoration.

Liq. ammon. acetatis, carbonate of ammonia, and chloric ether (20 days). Brandy, 6 oz., increased to 12 oz. on 8th day. Turpentine stupes. Sesquichloride of iron during convalescence.

*Pneumonia.—C. W., æt. 16. Cigar Maker. Adm. April 10. *Died* on April 14. In H. 4 days. Dr. Beale, not in case book. No history. On adm. face flushed, violent delirium, skin hot, no rash. P. 84, R. 20. Dulness on percussion, crepitation, and bronchial breathing over right base. Left base not examined. Unconsciousness. P. 140. Death. P.M.—Red hepatization of lower lobes of both lungs. Kidneys congested. Brain not examined.

Dilute nitro-muriatic acid, liq. cinchonæ and chloric ether. Brandy 24 ozs. Turpentine stupes.

*Pneumonia.—C. L., æt. 19. Servant. Adm. Mar. 31. Disch. May 20. In H. 50 days. *Recovery.* Dr. Johnson, vol. xvi, p. 159. Syphilis 12 months ago. Prev. ill. 14 days. Got wet. Shivering, headache, and dry cough. On. adm. dulness on percussion over left lung behind, from angle of scapula downwards with increased vocal vibration, bronchial breathing and bronchophony, and slight crepitus. P. 108, R. 26. On 16th day friction sound below left mamma and angle of left scapula (5 days). No expectoration throughout.

Carbonate of ammonia and chloric ether. Turpentine stupes to chest. Quinine. Iodide of Potassium. Brandy, 3 ozs. (11 days), then 4 ozs.

*Pneumonia.—J. C., æt. 36. Tailor. Adm. June 9. Disch. June 24. In H. 15 days. *Recovery.* Dr. Garrod, vol. 1, p. 132. For 9 years short-winded and subject to winter cough. Prev. ill. 4 days. Shivering, loss of appetite, pain in right side, cough, and dyspnœa. On 2nd day rusty sputa (11 days). On adm. dulness (14 days), increased vocal vibration, bronchial breathing (4 days), and crepitation over lower lobe of right lung. P. 114, R. 48. Returning crepitation on 7th day. Convalescent about 18th day.

Carbonate of ammonia gr. x. 6tis horis. Calomel and colocynth. Turpentine stupes. Brandy, 6 ozs. for 5 days, then 4 ozs.

*Pneumonia.—J. K., æt. 30. Porter. Adm. Mar. 12. Disch. April. 4. In H. 23 days. *Recovery.* Dr. Beale, vol. 6, p. 117. Prev. ill. 7 days. Commenced with pain under right nipple, and cough. On adm. dulness on percussion, and

fine crepitation over lower two-thirds of lower lobe of right lung. Sputa yellow, transparent, and viscid. Chlorides of urine very small in quantity.

Carbonate of ammonia and chloric ether (6 *days*). *Turpentine stupes. Quinine. E.L. to right side on* 14*th day. Brandy,* 4 *ozs.*

*Pneumonia.—J. C., æt. 41. Labourer. Adm. April 14. Disch. May 20. In H. 36 days. *Recovery.* Dr. Beale, vol. 6, p. 129. Cough all last winter. Prev. ill. 11 days. Shivering, loss of appetite, great prostration, cough, and shortness of breath, hæmoptysis diarrhœa, and vomiting. On adm. P. 108, R. 44. Dulness on percussion, and large crepitation over right side behind from spine of scapula downwards. Expectoration very copious, frothy, and rusty. Bronchial breathing from 14th to 28th day. Large crepitation over base of left lung on 32nd day. Slow convalescence.

Carbonate of ammonia, chloric ether, and liquor ammoniæ acetatis. Brandy, 18 *ozs. Turpentine stupes. Sesquichloride of iron during convalescence.*

*Pleuro-Pneumonia.—J. A., æt. 20. Compositor. Adm. May 14. Disch. May 27. In H. 13 days. *Recovery.* Dr. Johnson, vol. 16, p. 205. Prev. ill. 8 days. Got wet. Shivering, headache, and loss of appetite, and on 5th day pain in chest, cough, and expectoration of about 0j of blood. On adm. P. 68, R. 24. Dulness on percussion, bronchophony, diminished vocal vibration, and small crepitation over lower lobe of right lung. Expectoration frothy and untinged. No bronchial breathing. On 14th day crepitus over right base looser and more extensive, friction sound at angle of right scapula (8 days). Convalescent about 19th day

Carbonate of ammonia, liq. ammon. acetatis, chloric ether, and quinine, and opium for 6 *days. Then quinine, sulphuric acid and chloric ether. Turpentine stupes.*

Pleurisy.—Paul M., æt. 17. Employed in a mirror factory. Adm. Mar. 9. Disch. Ap. 20. In H. 42 days. *Recovery.* Dr. Johnson, vol. 15, p. 127. Prev. ill. 4 day. Shivering, headache, cough with slight expectoration, pain below left nipple. On adm. dulness over left lung as high as nipple in front, and to midway between spine and angle of scapula behind. Diminished vocal vibration and absence of breathing over dull space, friction sound in front. No œgophony. P. 100, R. 24. Tongue furred. Urine not albuminous. On 8th day dulness more extensive, no friction sound. On 27th day slight dulness at left base, respiratory sounds natural.

Liq. ammon. acetatis, carbonate of ammonia, and chloric ether. E.L. to left side on 13*th day. Quinine and sesquichloride of iron during convalescence.*

Acute Bronchitis.—C. R., æt. 40. Housemaid. Adm. April 21. Disch. May 9. In H. 18 days. *Recovery.* Dr. Johnson, vol. 16, p. 179. Pleurisy 6 years ago. Prev. ill. 14 days. On adm. crepitation over lower part of lungs behind. Very little constitutional disturbance. Convalescent about 21st day.

Carbonate of ammonia, chloric ether and liq. ammon. acetatis. Turpentine stupes. Quinine and acid.

*Acute Bronchitis.—W. P., æt. 62. Van-driver. Adm. March 23. *Died* on April 4. In H. 12 days. Dr. Beale, vol. vi., p. 124. Frequent attacks of gout during last 20 years. Grandfather gouty, but father not so. Seven months ago a severe attack of diarrhœa, lasting 3 weeks. Loss of appetite, flesh, and strength; vomiting, and sour eructations, 3 months. No abdominal tumour perceptible. On April 1st shivering, sore throat, pains in belly and limbs, loss of appetite. P. 100. Accompanied by severe bronchitis, under which he sank.—P.M.—Lungs emphysematous; tubercles in apices. Bronchial mucous

membrane considerably inflamed. Heart large. Left kidney dilated into cysts.

Compound jalap powders. Pepsine. On April 2, liq. ammoniæ, acetat., aromatic spts. ammonia, and chloric ether. Brandy, 4 oz., and turpentine stupes.

*Acute Bronchitis.—M. K., æt. 23. Servant. Adm. May 12. Disch. June 13. In H. 32 days. *Recovery.* Dr. Garrod, vol. 2, p. 113.—Amenorrhœa 5 months. Prev. ill. 3 days, commenced with shivering and pain in left side. Very low on adm., P. 124, R. 32. Rhonchus and sibilus all over lungs, fine crepitation at bases. Slight dullness over left base. Expectoration frothy and untinged. On 6th day P. 132. R. 36. Dullness over left lung behind, from spine of scapula downwards. Increased vocal vibration, and large crepitation. Expectoration more viscid. Convalescent about 20th day.

Aromatic spts. ammonia, liq. cinch., and chloric ether, afterwards changed for carbonate of ammonia gr x, 6tis horis. Turpentine stupes. Brandy, 6 oz. Quinine during convalescence.

*Acute Bronchitis.—B. L., æt. 16. Smith. Adm. June 1. Disch. June 13. In H. 12 days. *Recovery.* Dr. Beale, vol. 5, p. 176. Short-winded and subject to coughs. Slight cough and expectoration, 5 months. Never spat blood. Prev. ill. 14 days. On adm. high fever; rhonchus and sibilus all over chest; resonance normal. Expectoration scanty and mucilaginous.

Dilute nitric acid and chloric ether. Turpentine stupes.

*Bronchitis.—Bronzed Skin.—E.U., æt. 24. Cook. Adm. May 12. Disch. June 12. In H. 32 days. *Relieved.* Dr. Beale, vol. 6, p. 150. Has had cough and palpitation, and shortness of breath on exertion for some years. Prev. ill. 3 weeks. Pale, weak, and thin. Thyroid gland much enlarged. Brown patches with light interspaces on skin of chest, arms, hips, neck, and cheeks. Appetite bad. Cough and copious mucous expectoration. Crepitation over bases of lungs. R. 32, P. 100. Basic systolic bruit. Venous hum. in neck. Improved.

Sesquichloride of iron, hydrocyanic acid, and glycerine. Wine, 12 oz.

Bronchitis—L. S., 57. Married. Adm. March 5. Disch. May 16. In H. 72 days. *Much relieved.* Dr. Beale, vol. 5, p. 138. Bronchitis, and œdema of ankles; sloughing ulcer on left ankle.

Sesquichloride of iron, and inf. quassiæ. Morphia. Carrot and yeast poultices to ulcer.

Chronic Bronchitis.—C. C., æt 60. Adm. April 27. Disch. May 16. In H. 19 days. *Recovery.* Not reported in case book.

*Emphysema and Bronchitis.—Dropsy.—E. S., æt. 43. Cabman. Adm. May 14. *Died* on June 13. In H. 30 days. Dr. Johnson, vol. 15, p. 185.—Intemperate. D. T. 10 yrs. ago. 7 or 8 attacks of gout. Winter cough 9 yrs. Prev. ill. 1 month. Œdema of legs and ascites, 14 days. Face dusky. Urine scanty and albuminous. Rhonchus and crepitation over lungs. Expectoration copious, purulent, viscid. P. 116, R. 24. Great relief from puncture of legs. P.M.—Lungs adherent, emphysematous, congested. Heart, 18 oz. Right ventricle thick and firm, right auricle dilated, left ventricle flabby, mitral orifice large, valves healthy, kidneys conjested.

Carbonate of ammonia and chloric ether (4 days), then quinine and chloric ether. Turpentine stupes. Brandy. Chlorodyne.

*Emphysema and Bronchitis.—Dropsy.—Purpura.—M. F., æt. 47. Midwife. Adm. April 7. *Died* on April 13.—In H. 6 days. Dr. Johnson, vol. 16, p. 161. Cough and occasional dyspnœa, 10 yrs. Much worse during last 2 yrs. Anasarca 6 wks. Hæmatemesis 14 days ago. Purpuric eruption, chiefly on

back and legs, 1 wk. On adm. face livid and anxious. Considerable dyspnœa. General dropsy. Large purple patch, after gangrenous, on outer and upper part of right thigh. P. 144, R. 40. Rhonchus and sibilus over lungs. Pulsation in scrobiculus. No bruit, urine normal. P.M.—Heart, 17½ oz. Right cavities dilated, atheromatous patches on valves. Upper lobes of lungs adherent, containing scattered chalky masses. Liver everywhere adherent, 3 lbs., fatty.

Aromatic spts. of ammonia and sulphuric ether. Brandy.

Emphysema and Bronchitis.—Dropsy.—S. H., 51. Coach Spring Maker. Adm. June 1. Disch. June 13. In H. 12 days. *Much relieved.* Dr. Johnson, vol. 17, page 45. Not very temperate. Acute rheum. 12 yrs. ago. Rheumatic gout 9 yrs. ago. Winter cough, 4 yrs. Prev. ill. about a week. Œdema of legs. Urine slightly albuminous. Cough since last winter. Physical signs of emphysema. Rhonchus and sibilus over lungs. Convalescent from dropsy on 8th day after adm.

Carbonate of ammonia and chloric ether.

Emphysema and Bronchitis.—Dropsy.—J. K., æt. 36. Hatter. Adm. April 16. Disch. May 16. In H., 30 days. *Relieved.* Dr. Beale, vol. 5, p. 131. Always short-winded, and subject to cough; worse in winter. Œdema of legs 2 mos. On adm. general dropsy. Chest emphysematous. Mucous râles over it. Expectoration copious and frothy. Pulsation in scrobiculus, where a systolic bruit is heard. Urine 10 oz. in 24 hours; slightly albuminous, containing granular casts.

Carbonate of ammonia, chloric ether, and compound decoction of broom tops. 4tis horis.

*Emphysema and Bronchitis.—J. W., æt. Adm. May 20. *Died* on June 3. In H. 14 days. Dr. Garrod, vol. 2, p. 134. Subject to winter cough. On May 11 œdema of legs (9 days), after long standing. On adm. face dusky, cough, and profuse expectoration. Rhonchus of sibilus, mixed with crepitation all over lungs. Systolic bruit over apex of heart. Abdomen distended, tympanitic and fluctuating. No albumen in urine. Delirious 5 nights. Heart's action irregular. Death. P.M.—Fluid in abdomen and pleuræ, lungs congested and emphysematous. Aneurism of thoracic aorta as large as an orange, eroding bodies of 9th, 10th, 11th, and 12th dorsal vertebræ. Spleen, 2½ oz. Heart fatty; valves healthy.

Liq. ammon. acetatis, carbonate of ammonia, and chloric ether. Asafœtida.-Bitartrate of potass and jalap powder o. a. m. Brandy, 12 *oz.*

Emphysema and Bronchitis.—H. H., æt. 38. Laundress. Adm. April 29. Disch. June 24. In H. 56 days. *Unrelieved.* Dr. Johnson, vol. 17. p. 8. Long subject to winter cough. Prev. ill. 3 months. Cough and dyspnœa. Chest very resonant. Rhonchus and sibilus. Sputa abundant, purulent. Systolic bruit over heart. No dropsy or scrobicular pulsation. On 8th day after adm. diarrhœa (11 days).

Carbonate of ammonia and chloric ether and senega. Squills. Astringents and opium for diarrhœa. Turpentine stupes.

Emphysema and Bronchitis.—A. S., 54. Commissionaire. Adm. Mar. 28. Disch. May 11. In H. 44 days. *Relieved.* Dr. Johnson, vol. 16, p. 151. Syphilis 30 yrs. ago. Cough and shortness of breath 3 yrs. Physical signs of emphysema. Rhonchus all over chest. Expectoration, copious, muco-purulent. P. 102, R. 34. Heart sounds normal. On Ap. 6, sudden numbness of left hand, and absence of pulse in left radial artery for about 6 hours. Circulation restored by friction of arm. On April 10th œdema of left leg, left popliteal vein hard and tense. Was discharged at his own request. Leg still œdematous. Bronchitis improved.

Carbonate of ammonia, chloric ether and senega. Quinine, chloric ether, and dilute sulphuric acid. Acetate of potash. Quinine, sesquichloride of iron, and chloric ether.

Emphysema and Bronchitis.—H. S., æt. 32. Waiter. Adm. Ap. 21. Disch. May 18. In H. 27 days. *Much relieved.* Dr. Johnson, vol. 16, p. 175. Intemperate. Father died of phthisis. Winter cough 10 years. On adm. lips blue, face dusky, breathing difficult, cough and muco-purulent expectoration. Chest barrel-shaped and resonant, crepitation over lungs. Pulsation in scrobiculus. On 6th day after adm. P. 82, R. 28. Improved. Disch. at his own request.

Carbonate of ammonia, chloric ether, and senega (16 days), then quinine, sulphate of zinc, and dilute sulphuric acid. Turpentine stupes. Inhalation of creosote.

Chronic Bronchitis.—J. R., æt. 74. Porter. Adm. Ap. 10. Disch. May 16. In H. 36 days. *Recovery.* Dr. Beale, vol. 6, p. 148. Cough 3½ years. Prev. ill. 2 days. On adm. cough, dyspnœa, mucilaginous and frothy expectoration. Rhonchus and sibilus over chest. Slight hœmoptysis on May 4.

Carbonate of ammonia, chloric ether and senega. Cod liver oil. Turpentine stupes.

Phthisis.—J. P., æt. 40. Street Hawker. Adm. Mar. 11. Disch. Ap. 22. In H. 42 days. *Relieved.* Dr. Johnson, vol. 16, p. 147. Father, mother, brother, and sister died of phthisis. Prev. ill. 4 months. Cough and loss of flesh. Profuse night sweats; occasional vomiting. On adm. dulness on percussion over right apex. Crepitus and prolonged expiration under clavicle and over supra spinous fossa. Rhonchus and sibilus over right base. P. 104, R. 28.

Cod liver oil, quinine, sulphuric acid, paregoric, and chloric ether.

Phthisis.—T. B., æt. 41. Musician. Adm. Mar. 20. Disch. Ap. 8. In H. 19 days. Dr. Beale, vol. 6, p. 121. Seven months ago while playing trombone vomited (?) blood. Since then cough and expectoration, night sweats, and loss of flesh. On adm. dulness under left clavicle, inspiratory click, expiration harsh and prolonged.

Quinine, digitalis, and sulphuric acid. Cod-liver oil.

Phthisis.—J. B., æt. 29. House-painter. Adm. Mar. 27. Disch. June 6. In H. 72 days. *Unrelieved.* Dr. Garrod, vol. 2, p. 88. Distinct blue line on gums, colic about 4½ months ago. Father died of phthisis. Prev. ill. 4 mos., cough and loss of flesh, night sweats, slight hœmoptysis. Both lungs dull over upper third, left most so. Tubular breathing under left clavicle, crepitation behind. Sputa purulent. P. 84, R. 24. Urine afterwards albuminous.

Syrup of iodide of iron and cod-liver oil. Afterwards quinine, and sulphate of iron, and sulphuric acid.

Phthisis.—W. C., æt 28. Tailor. Adm. Ap. 9. Disch. June 3. In H. 55 days. *Unrelieved.* Dr. Johnson, vol. 15, p. 197. No hereditary predisposition. Prev. ill. about 10 months, ascribed to his getting wet through. Cough and expectoration, night sweats, loss of flesh. Diarrhœa 5 wks. On adm. crepitus all over right side. Liver tender and enlarged. Trace of albumen in urine. On May 8th dulness on percussion over greater part of left lung in front, here and under right clavicle, large crepitation. Bronchophony under clavicles, loudest on right side. Diarrhœa ceased 2 days ago. Occasional hœmoptysis while in hosp.

Krameria and logwood, then sulphate of copper and opium, and then acetate of lead and opium. Cod-liver oil.

Phthisis.—P. B., æt. 44. Excavator. Adm. Feb. 27. Disch. Ap. 4. In H. 36 days *Relieved.* Dr. Johnson, vol. 15, p. 111. No hereditary predisposition. Pneumonia (right side) 3 years ago. Since then, cough and expectoration, and occasional spitting of blood. Slight hœmoptysis, dyspnœa, and pain in chest 11 days before admission. On adm. dulness on percussion over right apex, and over left back from spine of scapula downwards. Large crepitation over right apex

and left base. Crepitation, tubular breathing, and prolonged expiration under left clavicle. Expectoration copious and purulent. P. 100.

Carbonate of ammonia, and chloric ether, and liq. ammoniæ acetatis (14 *days*). *Then quinine and dilute muriatic acid. Cod-liver oil.*

*Phthisis.—Hepatic Disease.—W. O'D., æt. 29. Clerk. Adm. Mar. 16. *Died* on June 11. In H. 87 days. Dr. Garrod, vol. 1, p. 68. Intemperate. In K. C. H. under Dr. Budd from Nov. 29, 1862, to Jan 5. Had been ill a month with lumbar pain, scanty urine, and dropsy of legs and belly. About 25th day after adm., dulness on percussion, and large crepitation over lower $\frac{2}{3}$ of lower lobe of right lung, and greenish viscid expectoration. Constant vomiting. Urine containing oily casts, and $\frac{1}{3}$ albumen. Highest pulse 120; highest resp. 40. Disch. relieved.

Hydrocyanic acid and mist. effervescens, quinine and dilute nitro-muriatic acid, creosote. Brandy 9 ozs.

On re-admission was very weak, P. 128, R. 36, ascites much increased, urine $\frac{1}{2}$ albumen, dulness and large crepitation over the whole of right chest, vomiting almost incessant. Had been getting worse about 11 days before. P.M.—Right lung universally adherent, tubercle scattered through upper lobe, small cavity in back of apex. Left lung emphysematous, containing scattered tubercle.

Carbonate of ammonia, liq. ammon. acetatis and ammon.-citrate of iron. Diuretics. Bitartrate of potass and jalap powder. Hydrocyanic acid, and creosote, to check vomiting. Brandy 6 ozs.

Laryngeal Phthisis.—W. S., æt. 17. Gardener. Adm. Ap. 7. Disch. Ap. 16. In H. 9 days. *Slightly relieved.* Dr. Johnson, vol. 15, p. 163. Father died of phthisis. Cough 6 mos. Hœmoptysis 5 mos. ago, followed by gradual loss of voice. Night sweats. Redness of epiglottis and vocal cords. Ulceration of left false vocal cord, also of right false vocal cord to a less extent.

Cod-liver oil, quinine, and sesquichloride of iron.

Laryngeal Ulceration.—Phthisis.—G. H., æt. 23. Law-writer. Adm. Ap. 25. *Died* on June 3. In H. 39 days. Dr. Johnson, vol. 15, p. 175. Had syphilis 5 years ago, no secondary symptoms. Mother died of phthisis. Huskiness 18 mos. Prev. ill. 6 mos. Gradual loss of voice, and dyspnœa. On adm. thin and pale. Epiglottis swollen. True and false cords ulcerated, especially on right side. Dulness on percussion, and feeble breathing over right apex. The expectoration became nummular and fœtid. Dulness under clavicle increased, and breathing became harsh and bronchial. P.M.—Lungs adherent and crammed with turbercles. Cavity as large as an orange, filled with pus at back of right apex. Extensive ulceration of vocal cords.

Iodide of potassium, and liq. hyd. bichlor. Afterwards cod-liver oil, quinine, and chloric ether. Locally, sol. A.N., steam and creosote inhalation. Leeches to throat.

Laryngeal Disease.—J. M., æt. 32. Butcher. Adm. May 13. Disch. June 6. In H. 24 days. *Relieved.* Dr. Johnson, vol. 17, p. 29. Voice husky 5 mos. Enlargement of glands in left side of neck, $2\frac{1}{2}$ yrs. Swelling of left upper vocal cord. Chest sounds normal.

Quinine and sesquichloride of iron. Sol. A.N. to interior of larynx.

Laryngeal Disease.—J. T., æt. 69. Clerk. Adm. May 2. Disch. May 9. In H. 7 days. *Unrelieved.* Dr. Johnson, vol. 17, p. 4. Prev. ill. 10 wks. Occasional loud snoring noise during inspiration, aggravated by speaking. Slight dyspnœa and dysphagia. No loss of voice. Epiglottis much depressed, so that interior of larynx could not be seen with laryngoscope.

Sal volatile, then sulphuric acid; morphia at night.

*Cancer of Epiglottis.—W. H., æt. 64. Street Newspaper-seller. Adm. Mar. 13. Disch. May 27. In H. 75 days. *Unrelieved.* Dr. Johnson, vol. 15, p. 131. No hereditary predisp., and no appearance of cancerous cachexia. Prev. ill. 7 mos. Commenced with difficulty in swallowing solids. Cannot now swallow anything without great difficulty. Epiglottis the size of a walnut, very hard, and ulcerated on surface. While in hospital the tumour became larger and more ulcerated.

Chlorate of potass and glycerine (28 *days*). *Then iodide of potassium and liq. morph. mur.*

Subacute Laryngitis.—J. E., æt. 32. Hair net Maker. Adm. Mar. 20. Disch. Ap. 22. In H. 33 days. *Relieved.* Dr. Johnson, vol. 15, p. 141. Was struck on thyroid cartilage 5 yrs. ago. After 1 wk. complete loss of voice (3 wks.) and huskiness since. Prev. ill. 3 wks. Hoarseness, pain in larynx, and dyspnœa. On adm. complete aphonia, urgent dyspnœa, and pain and tenderness of larynx. Congestion and swelling of mucous membrane over arytenoid cartilages, vocal cords close together and immoveable.

Leeches to throat, chloric ether mixture, steam inhalation. Afterwards sesquichloride of iron and quinine, and inhalation of vaporised solution of tannic acid.

*Pericarditis.—W. B., æt. 28. Clarionet Player. Adm. June 3. *Died* on June 13. In H. 10 days. Dr. Garrod, vol. 2, p. 153. Had dysentery 5 yrs. ago, during voyage home from India. Cough and expectoration, and pain in left shoulder, about 6 wks. Prev. ill. 2 days. Cough and severe dyspnœa. On adm. cardiac dulness slightly increased. Loud friction sound over base of heart. Dulness and fine crepitation over right side behind, up to spine of scapula, rhonchus in front. P. 120, R. 48. Expectoration frothy, untinged throughout. Motions pasty, pale, and very offensive. Appetite bad. Urine albuminous. On 8th day, bronchial breathing up to angle of right scapula. On 11th day, severe pain all over front of chest. Dulness on percussion. Friction sound very loud. Delirium. Death. Highest pulse 148; highest resp. 48. P.M.—About 0j of fluid in right pleura, lower lobes of lungs carnified. Pericardium and contents the size of a pumpkin. Lymph on heart rough and recent. Pericardial sac distended with jelly like lymph. Wt. of heart 19¼ ozs. Abscess the size of an orange in left lobe of liver, its roof formed by attenuated diaphragm and pericardium. No communication with pericardial sac. Ragged ulcers in large intestine and ileum. Kidneys congested.

Quinine and alkaline mixture (6 *days*). *Aromatic spts. ammonia, chloric ether and tinct. cardamom. Co.* (3 *days*). *Carbonate of ammonia, squills and seneca. Calomel and opium. Brandy, up to* 24 *ozs.*

*Pericarditis.—W. A., æt. 36. Commissionaire. Adm. May 9. *Died* on May 15. In H. 6 days. Dr. Johnson, vol. 17, p. 21. Pain and swelling of knees 3 wks. Prev. ill. 7 days. Commenced with shivering. On adm. P. 92, R. 36. Cardiac dulness slightly increased; faint friction; sound over heart; lungs normal. On May 11, loud friction; sound over heart; fine crepitus at bases of lungs; no dulness; sputa untinged and frothy. P. 108, R. 36. Trace of chlorides in urine. No albumen. On May 13, violent attacks of vomiting after food or medicine. Dulness and crepitus all over back of lungs up to spine of scapula. Delirium during night of 14th, and rapid sinking the next morning. P.M.—About a pint of serous fluid in pericardium. Heart and pericardium covered with recent lymph. Fluid in both pleura. Right lung carnified.

Quinine, sulphuric acid, paregoric, and chloric ether (2 *days*). *Carbonate of ammonia, bicarbonate of potass, and quinine and opium. Turpentine stupes. Brandy,* 6 *oz. on adm.,* 12 *oz. on May* 14*th.*

Cardiac Disease.—E. W., æt. 34. Formerly a Soldier. Adm. Ap. 6.

Disch. Ap. 25. In H. 19 days. *Relieved.* Dr. Johnson, vol. 16, p. 163. No acute rheum. Pain in chest, palpitation, and shortness of breath on exertion, 3 years. Temporary left hemiplegia some months before adm. Lately, noises in ears, cephalalgia, vertigo, and dimness of sight. Heart beating 2 in. below and ½ an inch to left of left nipple. Presystolic bruit. Loudest at apex. P. small, 74.

Sulphuric acid and sesquichloride of iron. Morphia at night. Belladonna plaster.

Cardiac Disease.—J. G., æt. 26. Gardener. Adm. Ap. 23. Disch. May 11. In H. 18 days. *Relieved.* Dr. Johnson, vol. 16, p. 191. Acute rheumatism 6 yrs. ago. Pain in chest; shortness of breath on exertion; palpitation and vertigo, 3 years. Œdema of legs and ascites 2½ yrs. ago (9 wks.). Œdema of legs 1 yr. ago. Prev. ill. 1 wk., with increased œdema of legs. On adm. slight œdema of legs. Heaving impulse; cardiac dulness increased; apex 2 in. below and to left of left nipple. Systolic bruit over apex. Bronchitis while in hosp.

Sesquichloride of iron and chloric ether (11 *days*). *Acetate of potash, ammonio-citrate of iron, and chloric ether* (3 *days*). *Then bicarbonate of potash, chloric ether, and carbonate of ammonia.*

Disease of Heart.—Epilepsy.—H. C., æt. 37. Governess. Adm. May 14. Disch. June 23. In H. 40 days. *Unrelieved.* Dr. Johnson, vol. 15, p. 193. Acute rheumatism 10 yrs. ago. Epileptic about 9 yrs. Catamenia irregular for some time. Anæmic. Systolic bruit loudest over apex; diastolic bruit heard over juncture of 3rd left costal cartilage with sternum. Dulness on percussion over left clavicle, and crepitation below it. Harsh, prolonged expiration below right clavicle. Scanty mucous expectoration. Pulse small. Nine fits while in hospital.

Carbonate of ammonia and chloric ether (21 *days*), *then quinine and sesquichloride of iron. Chloroform inhalation for epileptic fits.*

Cardiac Dropsy.—A. R., 53. Married. Adm. Feb. 5. Disch. Ap. 11. In H. 65 days. *Relieved.* Dr. Garrod, vol. 2, p. 32. Never had acute rheumatism. Palpitation of heart and occasional œdema of legs, 3 yrs. Prev. ill. 3 mos. Œdema of legs. Ulcers just above ankles. Venous pulse in neck. Pulsation in scrobiculus. Harsh diastolic bruit at base of heart. Pulse irregular and intermitting. Increased vocal vibration. Feeble breathing and dullness on percussion over back of right lung from angle of scapula downwards. Pain in chest. Headache. Severe attack of palpitation while in hospital. Urine slightly albuminous after Feb. 26.

Carbonate of ammonia, and ammon. citrate of iron. Iron and digitalis, separately and in combination.

*Paraplegia.—M. B., æt. 40. Married. Adm. Ap. 6. *Died* on May 15. In H. 39 days. Dr. Beale, vol. 6, p. 134. Gradual palsy of right leg, 10 years; palsy of left leg, 1 year. On adm. rigidity and contraction of hamstring muscles. No loss of sensation or reflex action. Palsy of bladder and sphincter ani. Sudden sinking on 38th day after adm. P.M.—Much pus and shreds of viscid purulent matter, both outside and inside the dura mater. No disease of the bones. The pus had destroyed the dura mater in various places, and had passed through. It had extended from the lower part of the cord as high up as the cervical region. Mere membranous shreds and much altered cord tissue with pus and the pia mater were all that remained of the lower six inches of the spinal cord. Three or four little scales of hard and, apparently, bony matter were found in the pia mater, corresponding to the disorganised portion of the cord. The origin and cause of the condition were not cleared up by the P.M.

Belladonna (3 *wks*), *iodide of potassium. Wine*, 6 *oz.*

Paraplegia.—H. T., æt. 38. Ship Rigger. Adm. Mar. 16. Disch. June 10. In H. 86 days. *Much relieved.* Dr. Garrod, vol. 2, p. 78. Had syphilis. Paraplegia 7 months ago. Loss of sensation; involuntary evacuations. Complete recovery in 3 weeks. Prev. ill. 2 months. Gradual paraplegia, not becoming complete, preceded by lumbar pain. Sensation impaired. Difficulty in retaining and passing water for 1 month after adm.

Tincture of conium. Sulphate of iron and quinine. Strychnia. Treatment much varied.

Paraplegia.—C. D., æt. 55. *Died* on June 9. In H. 24 days. Dr. Beale, vol. 6, p. 160. Much exposed to weather. Bronchitis 2 months ago. Œdema of legs 5 wks. Loss of motion and sensation from umbilicus downwards. Evacuations passed under him. Urine albuminous.

Iodide of potassium. Afterwards carbonate of ammonia.

Hemiplegia.—J. P., æt. 45. Street Hawker. Adm. Mar. 10. Disch. Ap. 4. In H. 35 days. *Relieved.* Dr. Johnson, vol. 16, p. 139. Had lived badly for some months before adm. Drunk for 2 days. On 3rd day (day bef adm.) sudden giddiness, headache, and loss of vision. Speech thick. Face drawn to the right. Loss of motor power in left arm and leg. Worse up to third day after adm. On discharge motor power and articulation nearly perfect.

Blister to nape of neck. Calomel and colocynth. Iodide of potassium and bark.

Hemiplegia.—B. D., æt. 67. Adm. June 12. *Died* on June 14. In H. 2 days. Dr. Beale, vol. 5, p. 190. Taken ill in street. Semi-comatose on admission. On day after adm. palsy and rigidity of right arm, and rigidity of right leg. Evacuations passed under her. Stertorous breathing. Death on 2nd day. No P.M.

Calomel. Aromatic spirits of ammonia.

Hemiplegia.—T. P., æt. 62. Labourer. Adm. Ap. 17. Disch. Ap. 29. In H. 12 days. *Recovery.* Dr. Johnson, vol. 16, p. 171.—Had been travelling on foot during last 3 weeks. Had lived badly during that time, and had been without food for 24 hours before adm. Fell down in street. On adm. speech thick. Loss of motion, impaired sensation, and no reflex action on left side. Tongue protruded towards left side. Left pupil dilated and insensible to light. Almost well on 3rd day after adm.

Quinine and sulphuric acid. Beef tea and brandy.

Hemiplegia.—T. W., æt. 48. Saddler. Adm Ap. 24. Disch. June 3. In H. 40 days. *Unimproved.* Dr. Garrod, vol. 1, p. 92. Prev. ill. 3 months (1 month after discharge from K.C.H.) Sudden insensibility lasting an hour, and left hemiplegia. On adm. slight motor power in right leg, none in arm. Sensation impaired. Childish. Motions and urine passed under him. Paralysis not improved.

Ammonio-citrate of iron and iodide of potassium. Asafœtida. Purgatives. Seton in neck.

Hemiplegia.—W. L., æt. 23. Sailor. Adm. Mar. 30. *Died* on June 19. In H. 81 days. Dr. Johnson, vol. 16, p. 123. Disch. from K. C. H. 3 wks. ago. Convalescent from *left* hemiplegia. Found insensible 2 days before adm. On recovery slight *right* hemiplegia with loss of sensation. Inability to speak or swallow. Sphincters paralysed. Trismus, can just protrude tip of tongue from mouth. The palsied limbs gradually became flexed, and the muscles very rigid. Trismus continued with slight remissions. Deglutition much improved after 10th day. Strength became much reduced by diarrhœa, and by 80th day he could hardly turn in bed, the head had become drawn back very far, and the muscles of the neck very rigid. Respiration became difficult, and he died apparently from asphyxia.

P.M.—White softening, with oily granule cells in both corpora striata and in pons.

Delirium Tremens.—Meningitis.—G. H., æt. 30. Beadle. Adm. May 28. *Died* on May 29. Dr. Garrod, vol. 1, p. 106. Intemperate. Two or three slight attacks before. Prev. ill. 3 days. Commenced on 3rd day after a drinking bout that had lasted a week, with muscular tremors and sleeplessness. Very noisy and talkative the next day. Spectral illusions on 3rd day. On adm. P. 120, pupils dilated. The delirium increased, he became violent, and died suddenly. P.M.—Slight serous effusion into and opacity of arachnoid, chiefly over anterior lobes. Heart hypertrophied, ventricles full of blood.

Tr. digitalis, ʒvij., *in three doses.*

Facial Palsy.—R. M., æt. 17. Adm. June 7. Disch. June 20. In H. 13 days. *Much relieved.* Dr. Garrod, vol. 2, p. 156. Chorea 15 yrs. ago. Acute rheumatism 10 mos. ago. Slept with the window open on night of 5th. The next morning he could not whistle or close right eye. Mouth drawn to left side. Tongue protruded towards the right. Recovery almost complete.

Blister kept open behind right ear. Syrup of iodide of iron.

*Palsy of 3rd, 4th, 5th, and 6th Cerebral Nerves, Right Side.—M. B., æt. 67. Widow. Adm. Mar. 3. *Died* on Ap. 12. In H. 40 days. Dr. Johnson, vol. 16, p. 133. Pain in back of head and left temple 12 mos. Anœsthesia of right half of face, rigidity of right masseter and temporal muscles, and loss of taste in right half of tongue, 4 mos. Ptosis of right eyelid, palsy of recti and loss of sight of right eye, 6 wks. On adm. loss of sensation in right side of face, right eye motionless, cornea insensible. No action of right temporal and masseter muscles. Pain in right side of face. Twitching of facial muscles. Death preceded by severe pain in head. P.M.—Pus beneath dura mater of inner part of right middle fossa of base of skull. A firm scirrhous growth occupied the right anterior fissure of cavernous sinus and extended into the body of sphenoid, posterior part of orbit and carotid canal. Adjacent bone carious.

Iodide of potassium. Morphia. Locally, sulphate of atropine, belladonna, aconitine ointment.

General Paralysis.—Bronchitis.—C. P., æt. 53. Shopman. Adm. May 16. Disch. May 27. In H. 11 days. *Relieved.* Dr. Beale, vol. 5, p. 162. A free drinker. Winter cough 6 yrs. Four attacks of delirium tremens during last 4 mos. Incontinence of urine, 4 mos. Numbness and loss of power in legs 2 mos. Gradual extension of paralysis to arms. Hands feeble, especially the right. Right pupil smaller than the left. Sibilus over lungs. Frothy expectoration.

Carbonate of ammonia, iodide of potassium, and ammonio-citrate of iron.

Amaurosis.—S. P., æt. 40. Valet. Adm. May 16. Disch. June 3. In H 18 days. Made O.P. Dr. Beale, vol. 5, p. 168. Had syphilis 14 yrs. ago followed by sec. symptoms. Gradual loss of sight of right eye, 5 mos. Left eye affected, 2 mos. Cannot see to write or read. Can distinguish large objects about 20 feet off.

Iodide of potassium. Blisters behind ears.

Delirium Tremens.—E. H., æt. 39. Needlewoman. Adm. June 9. Disch. June 13. In H. 4 days. *Recovery.* Dr. Beale, vol. 6, p. 180. Very intemperate. Drinking for 5 days before adm. Convalescent on day after adm.

Liq. ammon. acetatis and aromatic spirits of ammonia. Tr. Opii. m xxv. h s. s.

Delirium Tremens.—G. S., æt. 58. Adm. May 19. Disch. May 26. In H. 7 days. *Recovery.* Dr. Johnson, vol. 16, p. 213. Very intemperate. Epileptic 13 yrs. Frequent attacks of delirium tremens during last 12 yrs. Had been

drinking for 10 days before adm. Prev. ill. 2 days. Sleeplessness. Spectral illusions. No tremor. Tongue steady. Convalescent on 9th day.
Quinine and sulphuric acid. Opium. Purgatives.

Delirium Tremens.—E. M., æt. 40. Charwoman. Adm. June 23. Disch. June 29. In H. 6 days. *Recovery.* Dr. Beale. Erysipelas in face 6 yrs. ago. Has lately had trouble, and lived badly. Headache and sleeplessness for last few days. Slight tremor. P. 48.
Carbonate of ammonia and chloric ether. Opium. Purgatives.

Delirium Tremens.—B. D., æt. 32. Currier. Adm. Ap. 23. Disch. May 2. In H. 9 days. *Recovery.* Dr. Johnson, vol. 16, 185. Had been drinking for a fortnight. Sleeplessness, 1 wk. Tremors. Slight spectra. Two epileptic fits on 24th. Noisy on 26th and 27th. Slept on night of 27th. Rational on 28th. Free from tremor on 29th. Convalescent on May 1.
Purgatives. Opium. Quinine and acid. Brandy 6 ozs., and porter 0j.

Delirium Tremens.—J. H., æt. 52. Adm. Ap. 19. Disch. Ap. 29. In H. 10 days. *Recovery.* Dr. Johnson, vol. 15, p. 169. First attack 15 yrs. ago. Has had several since, the last 9 yrs. ago. Had been drinking 8 days before adm. Prev. ill. 2 days. Sleeplessness. Eyes ferrety. Considerable tremor. Slept on 4th night. Spectra at night till 9th day.
Liq. morph. mur. ʒss o.n. *On 4th day, Pil. Sapon. Co.* gr. x. *statim Tr. Opii.* m.xl *at night. Purgatives. Quinine and sulphuric acid.*

Epilepsy.—M. McM., æt. 65. Stone Sawyer. Adm. May 28. Disch. June 9. In H. 12 days. *Much relieved.* Dr. Johnson, vol. 16, p. 225. Brought in by police. Had fits before. Delirium 3 days. Slight bronchitis.
Opium. Quinine and sesquichloride of iron.

Epilepsy.—D. P., æt. 54. Waiter. Adm. June 24. Disch. June 26. In H. 2 days. *Relieved.* Dr. Garrod, vol. 2, p. 200. Intemperate. First fit on morning of adm., three fits afterwards.
Aromatic spts. of ammonia. Pil. Col. Co.

Epilepsy.— — S., æt. 60. ———. Adm. June 28. Disch. June 29. In H. 1 day. *Relieved.* Dr. Garrod.
Aromatic spts. of ammonia. Castor oil.

Epilepsy.—A. W., æt. 48. Charwoman. Adm. May 18. Disch. May 23. In H. 5 days. *Relieved.* Dr. Beale, vol. 6, p. 156. Epileptic 6 yrs. Had a fit 12 days ago, and a very severe one on morning of adm. Tongue not bitten. Fits less frequent during last 2 yrs.
Sulphate of zinc and hyoscyamus. Pil. Col. Comp.

Epilepsy.—S. C., æt. 24. Married. Adm. June 15. Disch. June 20. In H. 5 days. *Recovery.* Dr. Garrod, vol. 1, p. 140. During last 4½ yrs. has had 4 fits. Fell down in street on evening of adm. Convulsions lasted 6 hours. A slighter attack the next day. Coffee-ground vomiting.
Calomel. Sulphate of iron and sulphuric acid.

Chorea.—S. S., æt. 12. Adm. Feb. 12. Disch. May 30. In H. 90 days. *Recovery.* Dr. Garrod, vol. 1, p. 46. Two former attacks 3 yrs. ago and 11 mos. ago. Prev. ill. 30 days. Loss of motion, and sensation in right arm and leg. Choreic movements affecting the paralysed limbs least. Speech much affected. Tenderness over lower dorsal vertebræ. Could walk alone by 27th day after adm. No fright or cardiac disease.
Ammonio-citrate of iron. Asafœtida. Cod-liver oil and shower-baths after 46th day. Purgatives.

Chorea.—G. J., æt. 14. Errand-boy. Adm. Feb. 2. Disch. May 16. In

H. 102 days. *Relieved.* Dr. Garrod, vol. 2, p. 28. Frightened. Fourteen days afterwards choreic movements began in mouth and left side. The right side became affected towards the beginning of April, and the movements of the left side then became rather less.

Oxide of zinc. Sulphate of iron. Asafœtida. Strychnia. Conium. Quinine and sulphate of iron. Shower-baths.

Convulsions.—T. J., æt. 4. Adm. June 27. Disch. June 29. In H. 2 days. *Recovery.* Dr. Garrod.

Chorea.—E. A., æt. 14. Book-folder. Adm. Mar. 14. Disch. Ap. 29. In H. 44 days. *Recovery.* Dr. Johnson, vol. 15, p. 139. Had chorea 5½ yrs. ago, confined to left side. Speech much affected. Prev. ill. 3 wks. Pain in left leg, followed by choreic movements, then in left arm. Speech and deglutition natural. Right side scarcely at all affected. No fright or cardiac disease.

Quinine and sesquichloride of iron. Shower-baths.

Puerperal Mania.—M. McC., æt. 22. Married. Adm. June 5. Disch. June 16. In H. 11 days. *Unrelieved.* Dr. Beale. Confined 3 wks. ago. Prev. ill. 3 days. Violent delirium. Abscess in right shoulder. After evacuation of matter, the shoulder healed rapidly, and she slept more, but did not improve in other respects.

Carbonate of ammonia and chloric ether (3 days). Then quinine and chloric ether. Liq. morphia mur. Brandy and beef tea.

Dysphagia.—A. S., æt. 54. Tailor. Adm. May 26. Disch. May 30. In H. 4 days. *Recovery.* Dr. Johnson, vol. 15, p. 208. Prev. ill. 6 days.

Dilatation by bougies. Sesquichloride of iron and quinine.

*Cancer of Stomach.—G. R., æt. 46. Jeweller. Adm. Jan. 26. *Died* on Ap. 2. In H. 66 days. Dr. Johnson, vol. 16, p. 85. Prev. ill. 2 mos. Pain in epigastrium, and frequent vomiting after food. Sour risings and flatulence. Rapid wasting. On adm., tumour to right of umbilicus, moveable, painful and tender. Coffee-ground vomiting. P.M.—Cancer, the size of an orange, much ulcerated, involving ⅔ of pylorus. Omentum adherent to stomach. Intestines matted together.

Bismuth, magnesia, and dilute hydrocyanic acid. Opium. Cod liver oil.

Hæmatemesis.—S. S., æt. 21. Married. Adm. May 30. Disch. June 13. In H. 14 days. *Recovery.* Dr. Beale, vol. 5, p. 175. Hæmatemesis 1 qt. about 18 mos. ago. Pain after food 3 wks. Vomited about 1 pt. of clotted blood on morning of adm.

Ice. Gallic acid, dilute sulphuric acid, and tincture of opium. Quinine and sulphate of iron.

Hæmatemesis.—J. N., æt. 65. Married. Adm. May 9. Disch. June 20. In H. 42 days. *Relieved.* Dr. Beale, vol. 5, p. 158. Prev. ill. 1 mo. Pain and soreness of epigastrium. Vomiting. Hæmatemesis. Constipation.

Chloric ether, dilute hydrochloric and hydrocyanic acids. Ipecacuanha and rhubarb. Castor oil and warm water enema.

Hæmatemesis.—E. C., æt. 20. Servant. Adm. Mar. 25. Disch. Ap. 25. In H. 31 days. *Relieved.* Dr. Beale, vol. 5, p. 147. Hard worked of late. Loss of appetite, and occasional vomiting, 7 mos. Catamenia suppressed 4 mos. Hæmatemesis (about 1 qt.) a week ago. Tænia.

Hydrocyanic acid and bicarbonate of soda.

Dyspepsia.—M. A. P., æt. 18. Servant. Adm. Ap. 24. Disch. Ap. 29. In

H. 5 days. Dr. Garrod, vol 2, p. 105. In K. C. H. with similar symptoms about a year ago. Pain after food, nausea, flatulence, sour risings, and loss of appetite. Anæmia.

Sal volatile, ammonio-citrate of iron, and inf. of calumba.

Dyspepsia.—R. E., æt. 26. Artists' Colour-maker. Adm. Mar. 30. Dis. April 8. In H. 9 days. *Recovery.* Dr. Johnson, vol. 15, p. 101. Prev. ill. 9 days. Giddiness, pain in abdomen, and diarrhœa. Blood noticed in motions before adm. On adm. loss of appetite, flatulence, and pain after food.

Quinine and sulphuric acid.

Mesenteric Disease.—A. C., æt 9. Adm. Ap. 6. Disch. May 20. In H. 44 days. *Relieved.* Dr. Johnson, vol. 16, p. 165. Anasarca after scarlatina, 5 years ago. Has had dropsy twice since. Purged 3 or 4 times a day. Abdomen tympanitic and tender. No evidence of ascites. Liver large. Appetite at first bad, afterwards voracious.

Chlorate of potass and tincture of opium. Cod liver oil and syrup of iodide of iron.

English Cholera.—M. S., æt. 29. Charwoman. Adm. June 12. Disch. June 17. In H. 5 days. *Recovery.* Dr. Beale. Prev. ill. 2 days. Vomiting and purging. Pains in limbs. Loss of appetite. Tongue furred.

Castor oil and laudanum. Afterwards sal volatile, chloric ether, and tincture of bark.

Diarrhœa.—G. D., æt. 17. Porter. Adm. May 8. Dis. May 11. In H. 3 days. *Recovery.* Dr. Johnson, not in case book. Attacked suddenly on morning of admission with intense pain in abdomen, vomiting, and purging. When admitted was in a state of collapse One loose motion afterwards. Recovered quickly.

Brandy. Mist. camph.

Diarrhœa.—D. R., 28. Servant. Adm. May 13. Disch. May 23. In H. 10 days. *Recovery.* Dr. Johnson, vol. 17, p. 33. Prev. ill. 3 days. Pain in abdomen, purging and vomiting. P. 120. Tongue coated and tremulous. Sleepless.

Nitro-muriatic acid, liquor cinchonæ, and chloric ether. Liq. morph. Mur. at night.

Constipation.—J. A., æt. 22 Hairdresser. Adm. May 18. Disch. May 30. In H. 12 days. *Recovery.* Dr. Johnson, vol. 15, p. 191. Prev. ill. 2 days. Abdomen painful and tender. Vomiting. Constipation. Dulness on percussion over cæcum. Scybala brought away by injection.

Castor oil and warm water. Enemata. Opium. Leeches to abdomen on 21st. Quinine and sulphuric acid.

Constipation.—E. S., æt. 27. Labourer. Adm. May 8. Disch. May 23. In H. 15 days. *Recovery.* Dr. Johnson, vol. 15, p. 180. A similar attack 3 months before. Constipation. Pain in abdomen. Dulness on percussion, and tenderness over cæcum. Tongue thickly coated. Had been salivated before adm. Convalescent on 18th.

Turpentine stupes. Castor oil and warm water. Enemata. Hirud. vj. *to abdomen on May* 10. *Afterwards quinine and sulphuric acid.*

Constipation.—J. W., æt. 19. Servant. Adm. Mar. 28. Disch. June 25. In H. 89 days. *Recovery.* Dr. Beale, vol. p. Hard worked. An attack of constipation and pain in abdomen last summer. Prev. ill. 8 days. Febrile symptoms soon disappearing. Tongue foul. Bowels obstinately confined. Hysterical.

Colocynth, aloes, and calomel. Castor oil, enemata, croton oil, and colocynth. Sulphate of magnesia and tinct. of jalap. Podophyllin and colocynth. Large doses of sulphate of magnesia. Galvanism.

Gonorrhœal Rheumatism.—J. J., æt. 21. Adm. Ap. 6. Disch. June 10. In H. 65 days. *Relieved.* Dr. Johnson, vol. 15, p. 157. Gonorrhœa 1 wk. before adm. Pain in right hip and right heel.

Iodide of potassium, guaiacum, quinine, and sesquichloride of iron. Blisters to hip.

Rheumatism.—A. C., æt. 23. Blacksmith. Adm. Mar. 2. Disch. Ap. 4. In H. 33 days. *Recovery.* Dr. Johnson, vol. 15, p. 117. Prev. ill. 3 months. Shoulders, elbows, wrists, knees and left ankle affected before adm. Shoulders, knees, and left foot painful on adm. Gonorrhœa.

Iodide of potassium, bicarbonate of potash and bark. Strychnia and sesquichloride of iron.

Acute Rheumatism.—Pericarditis.—Acute Renal Dropsy.—T. T., æt. 16. Light Porter Adm. Mar 20. Disch. June 25. In H. 97 days. *Recovery.* Dr. Garrod, vol. 1, p. 75. First attack of acute rheumatism 12 years ago. Has had 4 attacks since. Prev. illness 4 days. Ankles and knees, shoulders, elbows, and hands successively attacked. On adm. right knee, elbow, and hand swelled and painful. Systolic bruit over apex of heart. P. 120, R. 40. On 8th day pain in præcordial region. On 9th day to and fro sound heard over heart. On 12th day dulness and bronchial breathing over right lung behind from angle of scapula downwards. On 15th day dulness, bronchial breathing, and œgophony over lower part of left lung. On 18th day diminished vocal vibration over left side behind, crepitus over left lower lobe, and viscid rusty expectoration. On 20th day, friction sound over base of right lung. On 23rd day, no friction sound audible, crepitus over bases, right base still dull. Expectoration scanty, and almost free from blood. Convalescent from rheumatism about 34th day. Œdema of legs on 31st day. Urine containing blood casts and ¼th albumen. Urine still contained a small quantity of blood on discharge.

Quinine and alkaline mixture (8 *days*). *Bark and carbonate of ammonia* (14 *days*). *Syr. Ferri Iodid* (10 *days*). *Liq. ammon. acetatis* (23 *days*). *Quinine and sulphate of iron* (9 *days*). *Liq. ammon., acetatis, and carbonate of ammonia* (5 *days*). *Then ammoniated tincture of guaiacum. Bitartrate of potass and jalap.*

Acute Rheumatism.—T. H., æt. 19. Labourer. Adm. Ap. 1. Disch. May 6. In H. 35 days *Recovery.* Dr. Garrod, vol. 1, p. 81. First attack. Prev. ill. 1 mo. Commenced in right knee; then in right knee, ankles, and wrists. Soft systolic bruit over apex of heart. Urine alkaline on adm. (Had been under treatment 3 wks.) On 5th day after adm., pain over heart, and to and fro sound (4 days). Duration of illness about 58 days.

Bicarbonate of potass and quinine. Morphia at night. Iodide of potassium added to mixture on 14th day after adm.

Acute Rheumatism.—C. F., æt. 36. Married. Adm. Mar. 7. Disch. Ap. 4, In H. 28 days. *Recovery.* Dr. Garrod, vol. 2, p. 66. First attack. Prev. ill. 7 days. Left knee and hip, right knee and hip, ankles, shoulders, elbows, and wrists affected in succession. Profuse sweating. Systolic bruit over apex of heart on adm. P. 88. No pain after 25th day.

Quinine and alkaline mixture (5 *days*). *Bark and carbonate of ammonia* (3 *days*). *Then quinine and alkaline mixture with iodide of potassium.*

Acute Rheumatism.—F. D., æt. 23. Butcher. Adm. Ap. 8. Went out on June 2. *Convalescent.* In H. 55 days. Dr. Beale, vol. 6, p. 136. Father, rheumatic. Three former attacks, the last 8 mos. ago. Prev. ill. 3 days. Shivering, headache, loss of appetite, pain and swelling of ankles. Was on the river all the day before. On adm., shoulders, wrists, knuckles, ankles and knees affected. Systolic bruit over apex of heart. P. 120, R. 36. On 6th day, heart's sounds muffled, cardiac dulness increased, severe pain in region of heart. On 16th day,

dullness and friction sound over lower part of right lung behind. No pain in joints after 44th day.

Liq. ammon. acetatis and bicarbonate of potass. Quinine and opium. Brandy. Leeches to præcordia on 11th day. Blisters to knees.

*Acute Rheumatism.—S. T., æt. 20. Servant. Adm. June 1. *Died* on June 11. In H. 10 days. Dr. Garrod, vol. 1, p. 122. First attack. Prev. ill. 3 days. Commenced in ankles. The next day the knees, shoulders, elbows, and hands were affected. On 4th day, joints slightly red, profuse sweating, thick creamy fur on tongue. P. 120, R. 48, to and fro sound over base of heart. On 7th day, to and fro sound over whole cardiac region, becoming less intense at apex after 9th day, but remaining very harsh and loud at base till death. On 11th day, dullness over bases of lungs. Delirious for two hours before death. P.M. —Slight serous effusion into each pleura. Pericardium adherent by recent lymph, easily broken down. Heart 17 ozs. Slight atheromatous deposit at commencement of aorta. Kidneys much congested.

Quinine and alkaline mixture. Brandy. Hirud. vj *to præcordia. Tr. opii* mxx, o. n., *after 11th day.*

*Acute Rheumatism.—M. S., æt. 18. Servant. Adm. June 3. *Died* on June 18. In H. 15 days. Dr. Beale, vol. 5. p. 182. First attack. Prev. ill. 7 days. Ankles, knees, wrists, and right shoulder attacked in succession. On adm., knees much swelled, other joints less so. Thick white fur on tongue. P. 100, R. 32. On 9th day, systolic bruit over heart. Diarrhœa after 9th day. On 18th day, slight dulness over right base. On 20th day, crepitation over right base, tympanitis, joints nearly well. On 23rd day delirium, exhaustion, evacuations passed under her. On 23rd day bronchial breathing over right base. Death. P M.—Base of right lung carnified.

Bicarbonate of potass, chloric ether, and ammonia. Afterwards chloric ether, liq. ammoniæ acetatis and logwood. Brandy up to 24 oz. Stupes to abdomen. E L. nuchæ.

Acute Rheumatism.—H. J., æt. 17. Servant. Adm. May 7. Disch. May 30. In H. 23 days. *Recovery.* Dr. Garrod, vol. 1, p. 98. First attack. Shivering and headache 7 days before adm. Had got wet through the day before. On 4th day the left ankle and shoulder became painful and swelled. On adm. (8th day) profuse sweating, tongue thickly coated, knees, shoulders, ankles, and right hand swelled and painful. Systolic bruit over heart, loudest at apex. P. 106. On 10th day, a friction sound over apex (4 days) No pain after 16th day.

Bicarbonate of potash and quinine (14 *days*). *Then quinine and sulphate of iron. Hirud.* vj *to præcordia on 10th day.*

Acute Rheumatism.—M. A., æt. 26. Servant. Adm. Ap. 29. Disch. May 16. In H. 17 days. *Recovery.* Dr. Garrod, vol. 1, p. 96. First attack. Prev. ill. 7 days. Knees, left foot and hand, and right shoulder swelled and painful. Præcordial pain 3 days. Profuse sweating. Tongue thickly furred. P. 88, R. 36. Basic systolic bruit over heart. Joints quite well on 4th day after adm. Left elbow afterwards painful.

Bicarbonate of potass and quinine (10 *days*). *Then sulphate of iron and quinine.*

Acute Rheumatism (?) Hysteria.—E. J., æt. 18. Servant. Adm. Ap. 15. Disch. May 19. In H. 34 days. *Recovery.* Dr. Garrod, vol. 2, p. 98. Hard worked. Prev. ill. 5 days. Shivering and prostration of strength followed after 2 days by pain and tenderness of ankles, headache, vomiting, and profuse sweating. On 5th day, shoulders, knees, and præcordial region painful and tender. On 7th day, P. 68, R. 36. great tenderness on pressure over præcordial region. On 12th day, no pain or tenderness anywhere. Appears quite well. Tenderness over dorsal vertebræ on 25th day. Herpes zoster on 33rd day.

Bicarbonate of potass and quinine. Asafœtida. Ammonio-citrate of iron and aromatic spts. of ammonia.

[*To be continued.*]

I.—CASES ILLUSTRATING THE FORMATION OF SO-CALLED "FALSE MEMBRANES," IN CONNECTION WITH THE IMMEDIATE COVERINGS OF THE BRAIN; WITH OBSERVATIONS.

II.—CASES OF SCROFULOUS DEPOSIT WITHIN THE SPINAL CANAL; WITH OBSERVATIONS.

III.—CASE OF LACERATION OF THE LOWER PART OF THE RIGHT INTERNAL JUGULAR VEIN, CAUSED BY FRACTURED PORTIONS OF CLAVICLE.

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Continued from page 91, *No. VI., Vol. II.*

Case 16.—"*False Membrane*" *in the Arachnoïd Cavity.*—*Scirrhous Ulceration of the Scalp.*—*Gangrene of the Lung.*

The patient was a woman, Sarah T. aged 46, who was admitted into St. George's Hospital, and died March 16th, 1844.*

History.—She had been suffering, for five years, with ulceration of the scalp; and when admitted into the hospital there was a large fungus-like ulcer of the part, with much sloughing, and exposure of the subjacent bone. Eventually much cough and bloody expectoration came on, and she died from extensive disease of the lungs.

Post-Mortem Examination.—*Thorax.*—Extensive Gangrene of the lung was found.

Cranium.—The scirrhous ulceration of the scalp was excessive, but the bone beneath was not diseased. The brain was natural in texture, though giving out much serum when cut into. There was an unusually large amount of fluid in the subarachnoïdean tissues, and in the lateral ventricles. Covering the left cerebral hemisphere a delicate and thin membrane was found; this was easily detached from the shining transparent arachnoïd beneath, and contained several dark "puncta," evidently owing to the presence of blood. Nothing like "bands" of adhesion were found between the opposed layers of the arachnoïd. No scirrhous ulceration existed in other parts of the body.

* Hospital Post-Mortem Book, 1844, No. 53.

Case 17.—*"False Membrane" in the Arachnoïd Cavity.—Old standing disease of the Corpus Striatum.—Hemiplegia.*

The patient was a woman, Zara S., aged 32, who was admitted into St. George's Hospital, April 29, 1845, and died September 2.*

History.—She had suffered from anasarca for seven months, and had been delivered of a child six weeks previous to admission. At the time of her confinement there was much bleeding, but the anasarca then disappeared. It again came on two weeks later, along with palpitation of, and pain at, the heart, to which, at a previous period she had been subject. When admitted, there was orthopnœa and physical evidence of consolidation of the lung; also symptoms of a weak, dilated heart. There was a slight amount of albumen in the urine, the sp. gr. of which was 1016. On the day after admission she was seized with a "fit," attended by total unconsciousness, general convulsive movements of the body, stertor with foaming at the mouth, and lividity of the face, followed by loss of power of the limbs on the r i g h t side of the body. On the day following she had recovered consciousness, but there was no movement of the right arm and leg; the sensibility of the skin of those parts was slightly affected. The speech was difficult, and the mouth drawn to the left side. She recovered the power of moving the right limbs to a certain extent; but was subsequently affected by much dyspnœa, with lividity of the face and delirium. From this she rallied, and, under the use of diuretics with cantharides and suitable stimulants, almost entirely regained power over the paralysed limbs. The dyspnœa increased, and also the albumen in the urine, to a great degree; she had hœmoptysis before death.

Post-Mortem Examination.—*Thorax.*—There was much fluid in the pleural sacs, and in the substance of the lungs, which were compressed. The heart was thinned as to its walls, and dilated as to its cavities; the mitral orifice was contracted by thickening of its valve-flaps.

Abdomen.—The liver was small and contracted, having a thickened and opaque capsule. The kidneys presented numbers of yellow striæ in their cortical parts. There was ulceration of the cervix uteri.

Cranium.—There was a large amount of serum in the subarachnoïdean tissues, and the arachnoïd membrane, generally, was opaque. The inner surface of the dura-mater, covering both hemispheres of the brain, was lined by a thin false mem-

* See Hospital Post-Mortem Book, 1845, No. 211.

brane of a red tinge. The dura-mater, forming the right portion of the tentorium cerabelli, was lined by false membrane in like manner. This false membrane contained a number of arborescent vessels, especially at the posterior part.

The cerebral ventricles were large, containing clear serous fluid. In the central part of the left corpus striatum there was a yellow spot of about the size of a sixpenny-piece, which was much softened. The brain-tissue immediately surrounding this was also softened, but not discoloured.

Case 18.—*"False Membrane" in the Arachnoïd Cavity.—Slight extravasation of Blood beneath the Arachnoïd Membrane.*

The patient was a man, Thomas B., aged 36, who was brought into St. George's Hospital, July 16, 1845, and died two days afterwards.*

History.—He had drunk very hard, and was admitted with pain in the head, and vertigo. For eight days he had been suffering from difficulty in getting sleep, which was attended by strange fancies; and for three nights, it was stated, that he had had no sleep at all. His illness began with feelings of prostration and pains in the limbs.

On the day of admission both pupils were noticed as being somewhat contracted, but acting well to light, and the face was flushed. The pulse was soft, though quick, and the tongue white in the centre. His manner was peculiarly abrupt and tremulous. On the day after admission he was much quieter, having slept better under the use of salines and hyoscyamus, with "bottled stout," as a drink.

Early on the subsequent day he was violently delirious, having gradually become very excited. Opium was given in the form of tincture (3i doses), but in vain. He rather suddenly became much quieter, began to breath in a very laboured manner and died.

Post-Mortem Examination.—*Cranium.*—The vessels of the scalp were much congested. The brain was firm, its cortical parts shewing many "bloody puncta;" the white parts being pale. The cerebral ventricles were somewhat dilated by slightly blood-tinged fluid. The whole of the parietal arachnoïd surface, covering the left central hemisphere, was invested by a thin tawny-coloured false membrane, easily capable of removal, but attached to the arachnoïd by fine and slender bands of adhesion. Here and there, in the false membrane, were blood-

* See Hospital Post-Mortem Book, 1845.

stained dark patches. The corresponding surface of the dura-mater (so called parietal arachnoïd) was perfectly healthy and smooth. Moreover, a small patch of recently effused blood was found beneath the visceral arachnoïd, covering the upper part of the right cerebral hemisphere, near to the longitudinal sinus.

Thorax and Abdomen.—The heart's chambers were somewhat dilated and the lungs congested; otherwise the organs of these cavities were not unnatural.

Case 19.—*"False Membrane" in the Arachnoïd Cavity.—Rupture of Diaphragm and Spleen.—Fracture of Bones of the Wrists after a fall.*

The patient was a man, James T., aged 24, who was admitted into St. George's Hospital, August 29, and died November 25, 1846.*

History.—He had fallen from a height, alighting upon the hands and fracturing the bones of the wrists. Sudden collapse came on, and this was followed by vomiting of blood, pain in the abdomen, and frequent attacks of syncope. Towards the latter end of life no pain was felt, but a sense of extreme prostration was experienced.

Post-Mortem Examination.—*Thorax and Abdomen.*—It was found that there had been rupture of the abdomen and spleen, and that, in connection therewith, abscesses had formed in the substance of the lungs and in the abdomen. *Cranium.*—A thin layer of fibrinous membrane was found lining the dura-mater over both sides of the brain.

Case 20.—*"False Membrane" in the Arachnoïd Cavity.—Scalp Wound.*

The patient was a man, James W., aged 32, who was admitted into the hospital Nov. 27, and died Dec. 11, 1846.†

History.—He was admitted in consequence of a scalp wound, one inch in length, which did not, however, expose the bone; this was owing to a fall whilst he was intoxicated. Headache followed; and subsequently œdema in the neighbourhood of the wound, which spread to the neck and throat. Delirium preceded death, which occurred whilst he was suddenly rising from his bed.

Post-Mortem Examination.—*Cranium.*—Covering the upper portion of the right cerebral hemisphere was a layer of

* See Hospital Post-Mortem Book, 1846, No. 243.
† See Hospital Post-Mortem Book, 1846, No. 254.

effused blood, and a layer of membrane, of a yellow colour, could be peeled off the dura-mater covering the same (the right) cerebral hemisphere. Also, on the opposite cerebral hemisphere a delicate false membrane was found in the arachnoïd cavity.

The other organs of the body presented nothing worthy of notice.

Case 21.—*"False Membrane" lining the Dura-mater.—Fever.*

The patient was a woman, Sarah T., aged 41, who was admitted into St. George's Hospital, August 2nd, and died on the 18th, 1848.*

History.—She was suffering from petechial fever, and had very decided cerebral symptoms. There was much want of sleep and also delirium.

Post-Mortem Examination.—The brain, on section, gave out a large quantity of serous fluid, which was abundant in the sub-arachnoïdean tissues and in the ventricles. On both sides of the brain the dura-mater was lined by a layer of soft membrane, evidently of recent formation, which had produced several adhesions between the opposed serous layers, chiefly at the upper and back parts of the brain.

Case 22.—*Membranous Layer of Blood in the Arachnoïd Cavity.*

The patient was a lady, Jessie D., the widow of a physician, who was brought into St. George's Hospital, with cholera, July 16, 1849, and died on the following day.†

History.—She was almost destitute, and, a few weeks before her admission, had sustained a severe blow on the head. For a month before admission she had had diarrhœa and sickness, and, at times, attacks of severe vomiting. She walked to the hospital, a distance of three or four miles, and when she arrived, the skin of her face was dusky, and the whole surface of the body cold. The pulse was about 85 per minute, but so feeble as scarcely to be perceptible. The breath was cool, and there was great thirst. The tongue was also quite cool, and much coated. The patient was, however, sensible, and complained much of want of sleep. Characteristic cholera-stools were passed by the bowels, and, in spite of treatment, she died.

Post-Mortem Examination.—*Abdomen.*—The glands of Peyer, in the intestines, were found to be unusually large, but nothing further of note was observed about the abdominal organs.

* See Hospital Post-Mortem Book, 1848, No. 176.
† See Hospital Post-Mortem Book, 1849, No. 149.

Cranium.—The brain-substance was rather "wet" on section, but, otherwise, presented nothing worthy of comment; over the cerebral hemisphere, however, on the right side, a thin lamina, or membrane, was found attached to, but easily removable from, the inner surface of the dura-mater. This was evidently formed of a layer of extravasated blood, and was tolerably recent; nevertheless, the colouring matter was beginning to assume a change, and the layer of blood was becoming transformed into a regular membrane.

No indications of injury from without, to the scalp or to cranial bones, was observed.

Case 23.—*"False Membrane" in the Arachnoïd Cavity.—Phthisis.*

The patient was a man, C. N., aged 21, who was brought into St. George's Hospital, February 24, and died March 9, 1858*

History.—He was admitted with pleurisy and phthisis. There were never any symptoms referable to the brain.

On Post-Mortem Examination, tubercles in the lungs and the results of old and recent pleurisy were found. The brain, itself, was healthy; but a thin layer of "false membrane" was met with lining the free surface of the dura-mater.

Case 24.—*Layer of Dense Membraneous Material, containing a cavity, attached to the inner surface of the Dura-mater.—Softening of the Central parts of the Brain.—Epilepsy.—Hemiplegia.—Tetanoïd Spasm.*

The patient was a woman, Mary H., aged 42, who was brought into St. George's Hospital, August 9th, 1850, and died on the second day following.†

History.—Five years previously she had been the subject of epileptic attacks, and, after one such attack, had lost the use of the left arm and leg. The leg had recovered to a considerable degree since, but the arm remained almost entirely without power of movement. Four days before admission she had a succession of from 25 to 30 severe convulsive attacks, and became unconscious. Subsequently muscular twitching came on in the limbs, and she was brought into the hospital partially, but not wholly, unconscious. The whole of the left side of the

* See Hospital Post-Mortem Book, 1858, No. — ?

† See Hospital Post-Mortem Book, 1850, No. 144.

body had lost all power of movement, and she was unable to speak or even to protrude the tongue. The face was flushed, the surface generally rather hot, and the pulse 120 per minute.

The bowels were well relieved by injections, and a blister applied to the nape of the neck.

On the day following she was rather more conscious, but the muscles at the back of the arms and legs were affected by clonic spasm, by which the body was thrown backwards in a succession of short tetanic paroxysms. Ice was applied to her head, and blood taken from the arm. Croton and castor oil were also administered, but no benefit followed treatment. Convulsions became more severe; the face more flushed; the evacuations were passed involuntarily; and coma came on before death.

Post-Mortem Examination.—*Thorax.*—The heart was soft in texture and flabby. The lungs were congested. *Abdomen.*—The kidneys were congested. *Cranium and spine.*—The spinal cord was congested generally. There was found extensive softening of the right corpus striatum, of the right optic thalamus, of the back of the fornix, of the under part of the corpus callosum, and of the central part of the posterior lobe of the right cerebral hemisphere. Moreover, on the right side of the brain, and corresponding to the right parietal eminence of the skull, a patch of yellow material, laminated in character, was found adherent to the inner surface of the dura-mater. This was of about the size of a crown-piece, and contained a kind of cavity, in which was a substance looking like silvery scales to the naked eye. This laminated substance could, without much difficulty, be peeled from the dura-mater, to which, however, it was somewhat firmly united. It was also, though very slightly, adherent to the visceral arachnoïd membrane corresponding. The arachnoïd membrane generally, over the right cerebral hemisphere, was more vascular than on the opposite side. There was a slight increase of the sub-arachnoïdean fluid, and the cerebral convolutions were somewhat flattened.

The cortical parts of the brain, generally, and also the cerebellum were very vascular.

[To be continued.]

II.—CASES OF SCROFULOUS DEPOSIT WITHIN THE SPINAL CANAL.

In this communication I propose to offer a series of cases, which have presented themselves during recent years at St. George's Hospital, showing the presence of scrofulous material within the spinal cavity, and illustrating the evil effects which are, for the most part, produced by such deposit upon the spinal cord. Many of the cases are associated with strumous disease of the vertebral elements; others, on the contrary, are not so associated. Again, it will be seen, that the effects upon the enclosed spinal cord vary very much, owing to the locality affected, the amount of scrofulous material deposited, its consistency, &c., &c. As I shall have the opportunity of relating several of these cases, I shall detail them in succession, and postpone such comments and inferences as may naturally arise from their consideration, until the whole have been related.

Case 1.—*Scrofulous deposit within the Theca Vertebralis.—Softening of the Spinal Cord.—Caries of the Vertebræ.—Hepatization of Lung.—Paralysis of the Legs and Arms, &c.*

The patient was a young man, Robert Glade, aged 20, who came under my care, at the St. George's and St. James's Dispensary, July 31st, 1856.

History.—The patient was one of a delicate family, some of whom had died of phthisis. His mother was hump-backed, owing, as it was said, to an injury to the back soon after the birth of the patient. He had experienced no blow or fall, and no severe illness in his lifetime; but, on the 11th of May, 1856, owing to pain in the right shoulder and a so-called "stiff neck" (which was thought to be, possibly, owing to rheumatism), from which he had suffered for the space of a month; he was obliged to give up his ordinary daily employment. Afterwards, he described the pain as going more into the neck and down the back, and as being much aggravated by coughing, or sneezing, or shaking himself, or when dressing, and when lying in bed he was quite unable to get up unassisted, owing to pain in the neck. His mother said he had latterly been "very nervous," and his memory had become somewhat defective. The hands were said to be generally cold, but not so the feet. Moreover, for one month there had been some difficulty in swallowing large morsels. Occasional headache was complained of, and at times vertigo, especially if the bowels were confined.

When first seen at the Dispensary, he was thin and spare, with cold hands, and in walking he supported the chin with his hand; he was also unable to approximate the chin to the breast owing to great and painful stiffness at the back of the neck. There was general tenderness on pressing the neck, which towards the lower part was somewhat full; and the right sterno-cleido mastoïd muscle was more tense and "taught" than its fellow. He complained of much "weakness" in both arms, but especially in the right one, so that he had a difficulty in lifting up anything of even very slight weight, or even to help himself at his meals; and the grasp with both hands, but especially with the right hand was very feeble. The legs were also weak in muscular power, and, at times, in walking, would "give way" at the knees; the right knee was too much inclined outwards. He always walked as if "with the greatest care." There was no cough, and there had been no hœmoptysis. He was wont to have palpitation of the heart, and the pulse was feeble: perspiration was apt to be abundant. Physical examination of the lungs and heart showed nothing wrong. There was no anasarca. The tongue was clean, the appetite good, but the bowels were often confined. The urine was free from albumen. The muscles of the face and also the pupils of the eyes were in a natural condition. The voice was unaffected.

On stripping the boy, it was found that there was curvature of the upper part of the dorsal region of the spinal column towards the right side; and great tenderness was produced by pressure at the middle of the dorsal as well as at the lower of the cervical part (as before mentioned) of the spinal column.

The treatment first adopted consisted in the application of the ung: antimon. potass tart. producing eruptions, about the spine, night and morning, and the use of quinine and cod liver oil, with occasional aperients. It was stated, August 12th, that the legs were weaker.

August 16th, the neck was described as being easier. He "could walk without holding up the chin," and there was no dysphagia. The legs were, however, getting more feeble, and both legs and thighs were stated (especially the right one) to "feel numb" when touched: this was the case also with the abdomen, as high up as the umbilicus. No starting or convulsive movements of the limbs existed, but often, on walking, the legs would "start out too far," and he would "take a longer step" than he expected. The legs were also described as often feeling "dead" in walking, especially the right one, and had of late been cold. The hands were cold, but there was no want of

sensibility of their surface. The ointment and medicines were continued. August 23rd, the legs were said to be much worse as regards numbness and want of sensibility, but he was "much better in himself," and stouter: he could raise himself in bed, and hold up his head better. There had been no spasmodic action of any of the limbs. The appetite was improved. He however complained of feeling pain occasionally across the chest, but there was no cough. The eruptions on the back had still not healed. Quinine and cod-liver oil were continued, and the iodide of potassium, with extract of gentian, given every night in addition.

September 6th, much the same on the whole. Pain was much complained of in the "left" shoulder-blade and arm; and also low down on the left side, above the crest of the ilium, which part was also painful on pressure; but no swelling existed at this region. He continued to pursue the same treatment until September 17th, when I got him admitted into St. George's Hospital, as the power in the legs had become so much more impaired. There was at that time decidedly less pain in the cervical region, even on pressure, but movement of the head in a lateral direction had become difficult. Blisters were, however, applied to the neck with decided relief, and later on a seton was inserted. The surgeon who was asked to examine the vertebræ could find no actual evidence of diseased bone, and the patient was placed under the influence, first of colchicum, and subsequently of the ammonio-citrate of iron. Under the latter he improved for a time, and became able to walk about the room; but the improvement did not last long, as at the beginning of December he was himself quite unable to extend his legs in bed, which, if extended by another, would, as a rule, gradually, though often quickly, become drawn up and flexed more or less on to the belly, the hamstring muscles becoming quite rigid. The sensibility of the skin of both legs, especially of the right one, was very deficient, and much reflex action of both legs could readily be produced by tickling the soles. It was also noticed that the left great toe was always extended, and frequent erections of the penis were complained of. Muscular power in the arms became more diminished. Towards the end of February, 1857, severe dyspnœa came on, and also some degree of cough; the paralysis both of arms and fingers becoming more complete. There was always more complete and decided "kicking up" (as the patient termed it) of the legs. The patient gradually sank; but three days before death had a severe attack of tightness across the chest and urgent dyspnœa, though very little expectoration. He however remained sensible to

the last, and no spasmodic movement of any kind came on before death, which occurred March 4th.

Post-Mortem Examination.—The body was somewhat emaciated. The spinous process of the last cervical vertebra was noticed as being very prominent.

Cranium.—The only thing noticeable about the cranial contents was that the cerebral veins were very turgid.

Spinal Column.—The posterior surfaces of the bodies of the last cervical and the three upper dorsal vertebræ were deprived of all ligamentous or fibrous covering, and the cartilage intervening between these vertebræ was almost quite destroyed, the opposed surfaces being carious. The anterior surfaces of the bodies of the five upper dorsal vertebræ were quite eroded and bathed in thick pultaceous white fluid and pus. It was also noticed that the yellow elastic tissue between the laminæ of the last cervical and first dorsal vertebræ was very remarkably softened and discoloured. On opening the spinal canal a large quantity of purulent fluid and of solid scrofulous deposit was found within the theca vertebralis, opposite the last cervical and first dorsal vertebræ, and this was especially so on the "left" side of the cord. By this deposit, moreover, the out-going roots of the spinal nerves on both sides, but chiefly those on the "left" side were closely surrounded by this deposit, which was very firmly adherent to the dura-mater at the posterior part of the canal. On examining the spinal cord itself, it was found that at the lower part of the cervical and upper portion of the dorsal region it was exceedingly softened, being, opposite the seventh cervical and the first dorsal vertebræ, almost diffluent.

Thorax.—The lungs, generally, were œdematous, and the lower portion of the right lung was hepatized. Much mucus existed in the bronchial tubes. The pleural cavities were natural, excepting that they were diminished by the bulging forward of the bag of pultaceous matter covering the anterior surfaces of the vertebræ.

Case 2.—*Scrofulous deposit occupying a part of the dorsal region of the Spinal Cord, which was also softened.*

The patient was a woman, Elizabeth D., aged 60, who was admitted into St. George's Hospital, August 3, 1831.

History.—For about eight weeks she had been complaining of pain in the back, dribbling away of the urine, and numbness of the legs. A few days after her symptoms first showed themselves there was a want of power in moving the right leg, and subsequently in moving the left leg; and afterwards the

sphincters lost their power, and the integuments of the back began to slough. She sank, and died August 21. Before admission into the Hospital she had been treated with strychnine, and been cupped and blistered. Spasmodic twitchings in the limbs, and a pricking sensation had been produced by the medicine, but no good results.

On Post-Mortem Examination, the lower part of the dorsal region of the spinal cord was found to be softened, and opposite the eleventh dorsal vertebra a mass of scrofulous deposit was found in the substance of the cord. The right half of the chord was more occupied than the left, but at no part was the deposit visible on the external surface of the cord.*

Case 3.—*Scrofulous material adherent to the outer surface of the Spinal Dura-Mater.—Spinal Cord healthy.*

The patient was a man, George P., aged 21, who was brought to St. George's Hospital, April 1, 1846.

History.—He had enjoyed general good health; but about two months previously he had experienced a feeling of constriction and pain across the chest, with a "shooting" to the back, which was followed in six weeks' time by a loss of sensation and of power of motion in the lower limbs, attended by a sense of coldness and numbness and by occasional twitchings in those limbs. When admitted, the general health was good, but there was pain between the shoulders when the vertebræ in that part were pressed, though no irregularity of the spinous processes, &c., existed; no headache was complained of; and there had never been any loss of consciousness. The bowels were much confined, and the motions, and the urine which was not albuminous, were voided with difficulty. After a time all the symptoms increased in intensity; and, by degrees, the arms, to a certain extent, lost sensation and power of motion. The urine also became alkaline and irritating. Perspiration of the skin was profuse about the head, neck, and shoulders; but below the latter the skin was always very dry. Great difficulty in breathing and expectoration of purulent matter came on, and bed sores formed before death, which occurred July 2nd.

Post-Mortem Examination.—Extensive grey hepatization of parts of the left lung, and much mucus in the bronchial tubes were met with, as also softened scrofulous deposit in the bronchial glands. A quantity of scrofulous material was found amidst the

* The preparation exists in St. George's Hospital Museum, Series viii, No. 140.

deep muscles of the back and around the 2nd, 3rd, and 4th dorsal vertebræ and the ribs, &c., which on their anterior surfaces were carious, and this deposit was continuous (by means of two of the intervertebral foramina, which were excessively enlarged) with a quantity of concrete deposit of the same nature within the spinal canal and adherent to the outer surfase of the dura-mater (but confined to the posterior part) of the dorsal region.* The spinal cord was at this part somewhat compressed, and was also softened by the pressure of the scrofulous material. The other parts of the spinal column, spinal cord, and membranes were healthy. The inner surface of the bladder was in a state of chronic inflammation. Moreover, the soft parts about the great trochanter, which was exposed by ulceration, contained an irregular deposit of new bone, quite apart from the original osseous structure of the trochanter.

Case 4.—*Scrofulous material occupying the Spinal Canal.—Pressure upon, and softening of, the Spinal Cord.—Caries of Vertebræ.*

The patient was a man, Michael M., æt. 33, who was admitted into St. George's Hospital, December 2, 1857, having previously been in the Charing Cross Hospital, and subsequently in the Middlesex Hospital.†

History.—He had had pain between the shoulders for 7 or 8 years, and for one year had noticed a projection at the "lower part of the back." Subsequently he lost all power of movement in the legs, and became an out-patient at Charing Cross, where counter-irritation was applied to the region of the curvature, which had the effect of diminishing the pain. The curvature increased, however, and a painful feeling of constriction around the abdomen was experienced, the walls of the abdomen being prominent and tense. Whilst in the Hospital a painful cough came on, and "tubular breathing" at the apices of both lungs was found on stethoscopical examination, along with dulness on percussion. Tonics with wine and good nourishment were given. The paralysis became more complete, and before death wandering of the mind came on. He gradually sank, and died January 30, 1858.

* See preparation in St. George's Hospital Museum, Series viii, No. 142.

† Mr. Shaw, of the Middlesex Hospital, in a note, dated February 6, 1858, informed me that when he saw this patient in that hospital, there was a "perceptible, but not great, projection of the spinous processes of the vertebræ in the dorsal region; and," he says, "I concluded that there was caries. The treatment consisted in painting the affected part with a solution of iodine, and giving tonics, with rest in the recumbent position."

Post-Mortem Examination.—*Thorax.*—The apices of both lungs contained masses of crude tubercle and vomicæ; numerous old pleuritic adhesions existed.

Cranium.—The bones of this cavity were natural. There was much soft fibrine among the sub-arachnoïdean tissues at the base of the brain, corresponding to the so-called circle of Willis, and especially around the optic nerves. The surfaces of the cerebral convolutions were flattened and dry, and the lateral ventricles were much distended with limpid fluid. The central parts of the brain, specially the septum of the ventricles, were much softened, but the fornix was not so. The anterior parts of the thalami optici were decidedly softened; and the vessels at the base of the brain, which to the naked eye appeared natural, were found by the microscope to contain numbers of fat globules within their parietes. *Spinal column.*—The bodies of the three lower dorsal vertebræ were found to be surrounded by a large mass of strumous material of about the consistence of putty, bound down by thickened pleura and sub-pleural tissue, and of this a quantity communicated with the interior of the spinal canal compressing the enclosed spinal cord, which at this part was considerably softened. The outer surfaces of the dorsal vertebræ, which were surrounded by scrofulous material, were much absorbed.

Abdomen.—The viscera of this cavity were natural.

Case 5.—*Scrofulous Abscess communicating with the interior of the Spinal Canal.—The Spinal Cord most probably pressed upon, but not softened.*

The patient was a woman, Mary B., aged 40, and was brought into St. George's Hospital, April 8, 1850.

History.—She had been an inmate of the hospital four years previously, owing to a fall on the back which produced very great pain, but no paralytic symptoms. Of this she recovered, and went on well until three months before her second admission, when she experienced much pain in her back, and had much cough. About eleven days before admission she quite suddenly lost power in both her legs, and when she came in there was evidently lateral curvature of the upper dorsal vertebræ, which were very painful. The stools became passed involuntarily, and a slough formed on the back exposing the sacrum; she sank and died, apparently from the extreme exhaustion owing to the discharge from the slough.

Post-Mortem Examination.—The lungs were found to contain a few old seams of scrofulous deposit, and the opposed layers

of pericardium were throughout adherent. In "the posterior mediastinum" and in front of several dorsal vertebræ (from the 3rd to the 8th), which were carious and softened on their anterior surfaces, was a large scrofulous abscess. The greater part of the body of the 7th dorsal vertebra was absorbed, and by reason also of the absorption of the cartilage between the 3rd and 4th dorsal vertebræ the abscess communicated with the spinal canal. Moreover, the lower part of the body of the 7th dorsal vertebra was tilted upwards and backwards, and, most probably, somewhat pressed upon, but had not softened or otherwise injured, the anterior columns of the spinal cord. The kidneys were congested. The other organs of the body were healthy.

Case 6.—*Scrofulous deposit within the Spinal Cavity and adherent to the outer surface of the Dura-Mater.—Spinal Cord pressed upon, but not softened.*

The patient was a boy, George S., aged 15, who was brought into St. George's Hospital, December 18, 1842.

History.—When admitted he complained of great pain at the back part of the neck, which was increased on motion, and he could not support the head when moved, without supporting it by the hands. There was numbness of the fingers, which increased; and soon afterwards the upper extremities became paralysed. Subsequently the lower limbs became also gradually paralysed, and incontinence of urine came on. Bed sores supervened, and before death, which took place October 14th, much difficulty in swallowing, and dysphagia, occurred.

Post-Mortem Examination.—There was extensive caries of several of the vertebræ, and also of the bones forming the base of the skull; and, corresponding to the 2nd, 3rd, and 4th dorsal vertebræ, was a quantity of scrofulous material, which was firmly adherent to the outer surface of the spinal dura-mater, and extended upwards, through the foramen-magnum, into the cranial cavity. It was found that the spinal cord was pressed upon, though not softened by, the deposit, which however was very firm and solid.* The other viscera of the body were free from disease.

* The preparation exists in the St. George's Hospital Museum, Series viii, No. 141.

Case 7.—*Scrofulous deposit occupying the cervical region of the Spinal Cord.—Similar deposit in the Brain.—Loss of power in the legs, arms, and sphincters, &c.*

The patient was a boy, Michael K., aged 13, who was brought into the hospital, May 2, 1849.

History.—He had for some weeks experienced a gradual loss of power in moving the legs and arms, and subsequently he had completely lost all control over the sphincters of the bladder and bowel. When admitted, much pain in the neck, increased on pressure, was complained of, and pain was produced by any movement of the head backward, but not by any lateral movement. There was also some degree of priapism. When the arms were extended by another person he could raise them, but he could not himself extend them. At a later period, the arms became firmly and permanently crossed over the chest, and great pain was occasioned by any attempt on the part of others to remove them; still later, rigidity of the sterno-cleido-mastoïd muscles on the right side came on; and for some days before death diarrhœa and bed-sores were established. Counter-irritation to the nape of the neck, in addition to other remedies, proved utterly ineffectual in affording relief, and he died September 10th, *the fæces having been observed to pass by the bladder.*

Post-Mortem Examination.—The lower part of the cervical region of the spinal cord (opposite the 5th, 6th, and 7th vertebræ) was much enlarged, and was occupied and rendered very solid by yellow scrofulous deposit in its entire thickness; the dura-mater and pia-mater corresponding to this portion of the cord were thickened and adherent to it.* Above the deposit, the spinal cord was very vascular, and also softened; but lower down it appeared healthy. A large mass of scrofulous deposit, of the size of a walnut, was moreover met with in the right optic thalamus, encroaching on the cavity of the third ventricle, and occupying a part of the substance of the left optic thalamus. The brain generally was rather softened and less vascular than natural; there was only a slight amount of sub-arachnoïdean fluid.

Thorax.—A slight amount of recently-deposited lymph was met with in the pleural sacs, and a small mass of cretaceous material surrounded by a fibrous capsule was found in one lung. In the left side of the pelvis was a sloughy cavity communicating with a slough at the back of the sacrum. This

* The preparation exists in St. George's Hospital Museum, Series viii, No. 139.

sloughy cavity also communicated with the cavity of the bladder at its neck, by an orifice containing phosphatic calculous matter, and also with the rectum by an opening, of the size of a probe, through its anterior wall. The bladder was very vascular.

[To be continued.]

III.—LACERATION OF THE LOWER PART OF THE RIGHT INTERNAL JUGULAR VEIN, CAUSED BY FRACTURED PORTIONS OF CLAVICLE.

Plate VIII.

This case I have thought worthy of being recorded, as it is in itself, I believe, of rare occurrence; and also because it calls to mind very strongly the case of the late Sir Robert Peel, the mode of whose lamentable death, most probably occasioned, at least in great part, by the wounding of a vein by a fractured clavicle (the left), was at the time looked upon as being almost, if not quite, unprecedented. The accident in this case, moreover, took place within a few months of the death of Sir Robert Peel.

Case.—A young man, aged 23, apparently in good health, was standing in Hyde Park, July 1, 1851, under some trees, in a thunderstorm. A rotten bough fell down from above him, and struck him against some hurdles. He died almost immediately afterwards, and was brought into our "dead-house" at St. George's Hospital quite dead. I examined the body, and found the following lesions:—*

There was great swelling and deformity about the lower part of the neck on both sides, and on opening the chest, and removing the sternum, &c., I found that both clavicles were extensively fractured, and that a very large amount of blood was extravasated about the portions of the fractured clavicles, and also beneath the cervical fasciæ. Fracture of the second rib on one side also existed, and in connection with this, there was a large quantity of extravasated blood among the pectoral muscles, and much also in both mediastina. On removing the fractured clavicles, I found that the lower part of the right internal jugular vein, at a point about one quarter of an inch above its junction with the subclavian to form the innominate vein, had been lacerated by depression of some fragments of the broken clavicle. The lacerated opening was oval in shape, its longest diameter being across the vessel, and was of such a size that it

* See St. George's Hospital Path. Museum, series vi, No. 172.

would about have admitted an ordinary sized pea. There was nothing like firm coagulum around or within the injured vein. The jugular vein of the opposite side was uninjured, and the subclavian veins on both sides were unaffected. On further examination some broken ribs were found on one side, and the lung corresponding was lacerated; moreover, the sternum was found to be fractured. Blood was also found extravasated between the peritoneum and the abdominal muscles. Nothing worthy of note was further observed in the body.

Observations. — Death was, no doubt, occasioned by hæmorrhage into the mediastina and the tissues of the neck, and among the muscles, &c. At least this must have contributed very materially to the fatal issue; and it is very clear that even if medical advice had been at hand at the moment of the accident, it must have been unavailing. How far the physical appearances presented by the neck and parts around the fractured clavicles resembled those in the case of Sir R. Peel, above alluded to, is of course entirely a matter of conjecture; but it is quite possible that examination of these parts after the accident, and before death, would have shown, as in Sir R. Peel's case, the existence of a "swelling" pulsating to the touch, and resembling a "diffused false aneurism." Of course the two cases so far differ, in that we have in the one case the lower part of the internal jugular vein lacerated, whereas in the other "it was supposed" that it was the subclavian vein which was injured.*

I do not see how it would be possible by ante-mortem appearances to diagnose laceration of the subclavian, from that of the internal jugular vein, at a point so low down as the injury in the present instance. As in Sir R. Peel's case, fracture of the ribs was found in this one also.

The accompanying illustration, Plate viii, which Dr. Westmacott made at my request, shows well the position, and size, &c., of the lacerated opening in the jugular vein. The proportions between this and the subclavian vein have been unfortunately not preserved, owing to the shrinking of the latter by reason of its not having been distended whilst immersed in spirits of wine.

* It will be remembered that there was no post-mortem examination of Sir R. Peel.

OF THE BRANCHING OF NERVE TRUNKS, AND OF THE SUBDIVISION OF THE INDIVIDUAL FIBRES COMPOSING THEM.

By Lionel S. Beale, M.B., F.R.S.

Plate IX.

Everyone who has endeavoured to follow by dissection the bundles of nerve-fibres which compose the large compound trunks, and trace them as they ramify in the branches, knows that it is not possible to track a particular strand of fibres for any distance, in consequence of the frequent changes which occur in its relation to other fibres entering into the formation of the compound trunk. A bundle of fibres may often be shewn to divide into two smaller bundles. Of these one may, perhaps, help to form a branch, while the other may continue onwards in the original trunk. Bundles of nerve-fibres often cross from one side of the trunk to the opposite, and very frequently, even by coarse dissection, a twisted arrangement may be demonstrated. In fact in all cases, although the constituent bundles of a trunk, and the constituent fibres of a bundle, appear to run parallel to one another, they really interlace very freely and cross one another frequently. It is therefore impossible to divide any nerve trunk longitudinally without cutting across a great many fibres; indeed it is probable that if a nerve trunk were simply slit for some distance in the longitudinal direction, all its fibres would be cut across as effectually as if the entire trunk had been severed transversely.

The arrangement of the individual nerve-fibres, figured in Plate ix, in the smaller, and even in the smallest trunks, is precisely the same as that of the bundles composing the larger trunks. The arrangement is constant, too, in all animals, and therefore seems to be essential or fundamental. I adduce it as a broad and most important fact in support of other arguments I have advanced in favour of the view that nervous organs invariably form complete circuits, and I hold that a circuit is a fundamental necessity for nervous action.

The arrangement presently to be described is at once explained, if there be outgoing and returning fibres in the same nerve trunk. It cannot be accounted for upon the hypothesis of the fibres being motor and sensitive, or afferent and efferent, in the same trunk, because the arrangement exists and is quite as striking in purely sensitive as it is in mixed nerves.

It is incumbent upon those who maintain that nerves terminate by distinct ends in tissues to offer some explanation of this peculiar arrangement, which seems quite inconsistent with the view they uphold. Neither Kölliker nor Kühne, nor any of the German observers who have lately been writing, seem to have

noticed this remarkable fact with regard to the course of nerve-fibres in trunks, although I have referred to it in my recent papers, and have drawn attention to it in my lectures for three or four years past. Dr. Arnold, of Heidelberg, has indeed lately described the arrangement as seen in the nerves of the Iris. (*Virchow's Archiv.* vol. 27, p. 345, 1863.) But it occurs in all nerves.

My friend Dr. Ciaccio has also delineated the arrangement as it exists in the nerves of the cornea (*Mic. Journal*, July, 1863). It is however not peculiar to the nerves of these textures, but is constant in all tissues. (See my paper "On the Ultimate Distribution of Nerves," *Archives of Medicine*, vol. iii., p. 236, plates xv., xvi., xvii, 1862. Also, *Phil. Trans.* 1860, Plate xxiii., Fig. 8.)

I will now briefly describe the arrangement as it may be observed in the nerves distributed to the pectoral muscle of the frog, and in those which supply the mucous membrane of the palate, the skin, and other parts.

The very first bundles resulting from the subdivision of the large trunk, are composed of fibres, some of which come from the nerve, while others seem to return to the nerve, or in other words the branches from the trunk contain fibres, which come from the centre, and fibres which pass towards the centre.

The lines drawn in Figs. 1 & 2, Plate ix, may stand for fine nerve-fibres, or bundles of nerve-fibres, for the arrangement exists in fine nerve-fibres, and in bundles composed of finer compound bundles. It is noticed with respect to the roots of the nerves as they pass from the cord. (See the papers of Mr. Lockhart Clarke, *Phil. Trans.*)

It is observed in the case of the fine nerve-fibres ramifying in the brain; it is found in all ganglia; it exists in the skin, in the fibrous tissue of the abdomen, and in the cornea of the frog, and is very easily demonstrated. The nerves distributed to the papillæ of the frog's tongue divide into two bundles at the base, which pass from the papillæ in opposite directions, while at the summit the fibres divide into very fine branches, which are connected with very numerous nuclei, and form a terminal plexus or network, which is situated at the summit of the papillæ, Plate ix, Fig. 5.

Fig. 3, Plate ix, is taken from a preparation of the pectoral muscle of the frog. It illustrates the manner in which nerves divide in the trunks, and the course taken by different fibres. Each fibre resulting from the division passes towards its distribution in different branches of the nerve.

Fig. 6 is also from the breast muscle of the frog, and represents two branches coming off from one trunk. Observe the

fibres dividing at *a*, *b*. The arrangement is shown more distinctly in Fig. 2, and I have placed arrows in the direction in which I conceive the nerve current to be passing. The arrangement is such that the currents pass in opposite directions in the fibres which are contiguous.

Fig. 4 is from the mylohoid of the hyla, and represents some of the finest nerve-fibres visible. It is magnified 1800 diameters.

It has been shown that a crossing of the nerve-fibres in trunks occurs, that the passage of nerves from the same bundle in opposite directions is constant in all tissues, and in all animals, and it has been conclusively proved, at least in many tissues, that such an arrangement is associated with the existence of a most intricate terminal network or plexus, composed of very minute fibres. Is it not therefore almost certain that in muscle and in all tissues a similar terminal network or plexus exists? If not, how is the peculiarity to be explained? If the nerves terminated in free extremities they would surely pass to their destination by the shortest route. The argument seems conclusive even in the absence of all demonstration. No one would be so rash as to assume that such an arrangement as that figured is purposeless and accidental. From its constancy it is most certain that it is essential, and from the fact that it exists in the case of old fibres, fully formed fibres, and in the youngest fibres, it is certain that the arrangement is necessary to the very nature of nerve distribution.

The facts described in this paper give support to my view with regard to the existence of nerve circuits—a view which is fully justified by the evidence given in former papers. How the arrangement is brought about shall be fully discussed in another communication. My paper, on the structure and formation of the so-called a-polar, uni-polar, and bi-polar nerve-cells of the frog, (*Phil. Trans.* 1863,) which will be published in a month, contains numerous observations which strongly support this conclusion, but from another point of view.

The general inference I deduce from a careful examination of the course taken by nerve-fibres in the largest and smallest nerve trunks, in bundles of nerves ramifying in nerve-centres and in peripheral parts is this:—That with regard to every nerve-cell, central and peripheral, there are—

1. Fibres, or at least a fibre, passing to the cell.
2. Fibres, or at least a fibre, passing from the cell.
3. Commissural fibres, passing between different cells and connecting with each other certain cells and sets of cells, which concur in their action.

In Fig. 2, Plate ix, *a* and *d* may be commissural fibres, *b* may be a fibre passing from, and *c* a fibre passing towards a central nerve-cell.

RECENT CONTRIBUTIONS TO THE STUDY OF CONTINUED FEVER.

No. I.

At the last gathering of the Social Science Association at Edinburgh, under the Presidency of Lord Brougham, Professor Christison delivered a very important address on Public Health, in which he discussed all the views advanced at different times upon the operation of external agencies in the production and spread of diseases of zymotic type, and expressed his opinion as to how far we might hope to prevent such diseases, or limit their spread, by the removal of such causes as dirt, defective drainage, bad air, overcrowding, &c.

An address, on such a subject from so philosophical a thinker, and so experienced a physician as Dr. Christison, naturally attracted a large amount of attention and discussion.

As our readers have doubtless already read that address elsewhere, we shall assume that they are conversant with the facts and reasoning advanced by Dr. Christison, but we have thought it well to call attention to it by way of preface to the papers analysed in this article.

En passant we may remark, however, that Dr. Christison's opinions seem to have undergone some modification since 1846, for, in the July number of the *Edinburgh Medical Journal* of that year, is a paper by him on "Typhoid Fever apparently originating in local miasma." The epidemic, he there describes, was so limited in its sphere of action, and virulent within the circle of its influence as to have given rise to suspicions of poisoning, although it really depended upon the disturbance of a soil which had been for a long time impregnated with the products of sewage.

The statistical method of investigation applied to questions of general health is open to many fallacies, among which may be adduced the stringing together under the category of "Typhus" all the continued fevers of this country, and, similarly, under the single term of "foul air" comprehending all kinds of foul air, produced by many and different agents.

Into the etiology of true typhus we do not propose to enter specially, further than to remark, that, in its prevalence, it has partaken more of an epidemic than an endemic character; that it proves eminently contagious when it has originated, is allowed on all hands; that there are good grounds for the belief that it can be generated by the overcrowding of human beings, debilitated and enervated by starvation, during the prevalence

of cold weather, but that its spontaneous origin (and the same may be said of typhoid fever) cannot yet be considered a settled point.*

From the last Army Medical Blue Book for 1861, just published, we have extracted the following observations from the various papers on fever.

Professor Christison holds the theory that diseases change their type, and that the generation and spread of these have some reference to epidemic constitutions of the season prevailing at different times.

Deputy Inspector-General Lawson's paper is upon the influence of "Pandemic causes" in the production of fever. In elucidating this subject he has laid under contribution the Army Statistical Returns of Sickness and Mortality, ranging, in time, from 1817 to 1856, and, in space, extending from Ceylon to Jamaica in longitude, and from Great Britain to the Cape of Good Hope in latitude.

Dr. Marston, in his report on fever, says:—

"It is highly probable that some atmospheric conditions 'beyond our ken' enter into the creation and spread of the fever poison. If within the limited area presented by a regiment or garrison we can more leisurely and exactly trace the steps of progression of a disorder, it must not be lost sight of that there are other causes and influences at work, upon a far larger scale—waves of diffused influence."

This, then, is the subject of Mr. Lawson's paper, and, from an examination of the data afforded by the official returns, he is enabled to trace the curious regularity "and progression of the oscillations of febrile disease at so many points on the earth's surface, far removed from each other, and embracing the continued, remittent, and yellow forms of the disease, as to leave no doubt of the operation of some cause equally extensively." . .

"The facts detailed establish conclusively the occurrence, between 1817 and 1836,† of a series of oscillations of febrile disease, following each other with amazing regularity. These have been attributed to a cause or influence which, from its extent and progressive character, has been designated a

* In the case of the "Schah Gehald," wherein it was asserted that "typhus" had been spontaneously generated among its living cargo, under circumstances parallel with those stated in the text, this has not been quite substantiated, because there was an absence of proof that none of the passengers or crew were affected at the date of their first embarkation.

† At the end of this paper the reader will find such a brief summary of the facts adduced by Deputy Inspector-General Lawson as will serve to indicate the manner in which he has worked out his subject.

'pandemic wave,' the term epidemic referring rather to a single form of disease affecting a limited space." . . .

"What the nature of this pandemic wave and its mode of action may be, it is difficult to say; but certain deductions may be made with advantage from the facts concerning it set forth above. Of these the most striking is the constant course in the Atlantic and western part of the Indian Ocean, from south or south-east to north or north-west. This can take place in virtue of some law of nature only, and the situation of the magnetic poles immediately suggests itself as being in some way connected with it; but of this there is not at present any proof so far as I am aware." . . .

"Another point of more immediate importance, as bearing on some of the medical opinions of the day, has been alluded to several times in the course of this paper, viz., the occurrence of every form of fever under the operation of the same pandemic wave, yellow fever, remittent, intermittent, and continued fever, and plague, having prevailed in the localities in which they were endemic as the wave passed over them."

"As it appears that in different countries different forms of fever prevailed, under the same general influence, it must be admitted that the pandemic cause determines the frequency and severity, rather than the particular form of the fever, which, there are many reasons to conclude, is more intimately connected with the local circumstances at the time."

"Sometimes, it is true, one of these forms invaded the territory of another for a while, and such intrusions have given rise to some of the most embittered controversies in the history of medicine. The principles here developed will place the evidence in connection with these unusual irruptions of disease in a new light, and contribute to reconcile the opposite opinions regarding them, giving us a basis to reason on epidemics better suited to the time, and the advance of our knowledge of the causes of disease, than that on which the subject was contested by Pym and Bancroft fifty years ago." . . .

"It is an interesting subject of inquiry whether the pandemic cause acts on the system, rendering that more susceptible of the influence of the ordinary causes of disease, or on these themselves, giving origin to a more concentrated, or a different kind of poison, which generates a more virulent, or a new form of disease; or whether it affect both, increasing the activity, or altering the kind of the causes, and enhancing the liability of the body to be affected by them. Our information on these points is still very limited and unsatisfactory. I have observed that prior to, and during the prevalence of, an epidemic of

yellow fever, the urine contained an unusual quantity of urrhodine, giving a deep reddish-brown when treated with nitric acid and set aside for some hours. Something similar has been observed with regard to cholera. These tend to show that the body itself is affected by the pandemic cause; but the observations require to be repeated, before much weight can be attached to them."

"The outbreaks of epidemic disease have been referred immediately by some to the rapid generation and diffusion of myriads of miscroscopic animals, and by others to a similar production of vegetable organisms. The evidence in support of either is very defective. It may be well to keep the possibility of such things occurring in view, while we cannot be too cautious in admitting their efficiency, without adequate and irrefragable proof."

"In the above remarks, the influence of the pandemic wave in producing fever only has been examined, but it is obvious in many other affections, of which cholera, diarrhœa, dysentery, and scurvy are the most important. Even consumption and ulcers vary so much from time to time in frequency, as to suggest that they are similarly affected."

Mr. Hanbury, Surgeon to the 33rd Regiment, describes his experience of typhoid fever in India, from a sanitary point of view.

The clinical and anatomical characters of the disease, illustrated in the cases he details, were, in his experience, of an unusual description. He indicates how this particular form of fever might easily have been classed under the head of the remittent fever of India, occurring, as the cases did, during the autumn months of the year, at a time when cases of intermittent and remittent types of fever were to be expected, and were actually present, and where the symptoms presented by many of them were suggestive of those diseases rather than of typhoid fever, which the post-mortem evidence proved them to have been. He traces the rise, prevalence, and diappearance of the disease in connection and relation with local sanitary causes.

In 1859 the garrison of Malta numbered 6,744 men, and 1,196 cases of fever (of all types) were treated, with a mortality of 41. Dr. Marston takes this as his text, upon which he relates his whole experience and observation of this class of disease.

The fevers considered are:—1. Typhoid; 2. Gastric—Remittent, and Bilious Fevers; 3. The three to five day fever of the Mediterranean.

Typhoid fever was the cause of the mortality in the garrison of Malta in 1859, and with this he commences. He considers the question of the non-identity of the typhus and typhoid types to be settled. He traces the introduction of typhus fever into Malta upon two occasions,—during the Crimean war, and in the spring of 1861, by the crew of the Egyptian vessel "Schah Gehald," which had attained such a morbid notoriety at Liverpool. The disease proved eminently contagious, but spread within an extremely limited range at Malta. He discusses, at some length, the etiology of typhoid fever, and its relations to other miasmatic diseases. He endeavours to prove that Dr. William Budd's and Professor von Geitl's theory, as to the spread of this disease by contagion only, and particularly through the infectious properties of the excreta of affected individuals, cannot include *all* the facts.*

Dr. Marston expresses some hesitation in the acceptance of these doctrines.

"We are in a condition to say that no one has hitherto produced any organism without the agency of pre-existing germs, and, conversely, that wherever such production has been the subject of observation and experiment, the conditions permitting the presence of such germs have never been absent; but it is to beg the point at issue, to say that the generation of typhoid fever rests upon the same basis as this, and that the impossibility of tracing the lineage is of no more force against propagation by continuous succession in the one case than the other. For contagionists to assert that what is not impossible may have taken place, is a secure way of guarding their theory, which would be only justifiable if there were the enormous preponderance of facts upon their side, such as we possess with regard to the transmitted descent of animals and vegetables. The numerous occurrences will remain unexplained, wherein intestinal fever has suddenly appeared in a limited and previously healthy locality, concurrently with a foul state of sewage, &c.—the progress of the disease ceasing with the removal of this, and the most careful inquiries having failed to trace the arrival or presence of an infected person from without

* As is well known, Dr. Wm. Budd has, in some very able papers recently published, not only endeavoured to establish the general contagious nature of this disease, but to fix the contagious and infectious properties upon the excreta from affected individuals, classing typhoid, cholera, and yellow fever in the same category, viz., ground infecting poisons. Dr. Budd is a total disbeliever in the spontaneous origin of this intestinal fever. He says that, to assert this upon the merely negative evidence of our inability to trace the continuity of the succession in this instance, we might, equally, hold the spontaneous generation of animals and plants.

—if the occasional spontaneous origin of the disease be not received."

In tracing the origin and course of the epidemic in Malta, Dr. M. appears to take the following grounds:—

1st. Some wave of diffused influence, or unknown atmospheric conditions which enter into the creation and spread of the fever poison.

2nd. A concurrence of local causes, such as impure drainage and sewage, giving use to fœcal odours and the products of animal decomposition, at a time coincident with such barometrical and thermometrical states of the air as fit it for the reception, absorption, and subsequent deposition of miasmata and malaria.

3rd. Contagion, whether arising from intercourse with affected individuals or exposure to excretions from their bodies.

That these influences may vary in the importance of the position they occupy, as causes of the spread of the disease in different times and places, is very probable. In the Malta epidemic the local causes seemed to hold the most prominent, and the personal causes a subordinate position, in this respect. In a small village this may be reversed, and contagion may come to occupy the first place. There can be little doubt that fever may spread in more than one way, and there is no more curious or important study than the varying combination of the epidemic, endemic, and contagious influences. Nay, further, the modification of fever (any form) by local causes is well shown by what Sir James McGregor says of plague, in the different localities of Egypt—sometimes taking on malarious, at other times the inflammatory type, &c.

From the facts detailed, he shews, that the fever appeared in one new barrack shortly after the opening of some sewers, and that, generally, the amount of the disease, and its severity, had an intimate connection with the inferiority (in a sanitary point of view) of the barracks occupied. And, on the other hand, he points out that in some forts, remote from barrack and town sewage, the disease was for a long time absent, and was very limited when it did appear.

He sums up the result of his observations on the causes of enteric fever in the following propositions:—

1. That typhoid fever is endemic rather than epidemic; when the latter, it is often remarkably circumscribed within one or more limited spheres.

2. That it is, in all probability, contagious; but that this is inadequate to explain all its outbreaks, and that contagion is to

be held in the light of a subordinate rather than a primary and principal cause of its spread.

3. That typhoid fever is, probably, etiologically allied to other diseases, such as gastro-enteric ones, having either a different or allied patheology.

4. That, in its origin and spread, it has an intimate connection with bad drainage and fœcal fermentation.

5. That the products of such fermentation may be varied in many ways, and that so, in all probability, they may also give rise to diseases differing from typhoid fever.

6. That enteric or typhoid fever is probably capable of a spontaneous origin, as opposed to that of continuous succession from the bodies of individuals under its influence.

7. That it ordinarily commences about autumn, and reaches its acme of prevalence soon after that season, continuing through the winter in mild climates, but ceasing towards the spring.

8. It is probable that the character and constitution of the season exert some influence. Qy. It has appeared in Malta more than once, after hot and dry summers, with the first autumnal rains.

He then analyses the different symptoms presented by the patients during the course of the disease.

Upon the subject of exanthem, he contrasts the differences between the typhus and typhoid eruptions, and adduces some cases in which purpuric or petechial spots have appeared with the "rose" papules, characteristic of typhoid fever; but such petechial spots have not pursued the gradual and continuous course of the typhus eruption, and are, he thinks, only to be considered as accidental to the stagnation of the blood in the capillaries, the impaired contractility of the skin, and the asthenic condition of the patient. In such cases the eruption, as a whole, consisted of the characteristic effaceable "rose" papules of typhoid fever, which came out and passed off in successive crops.

Vesicles sometimes, though excessively rarely, appear upon the summits of the spots. The medical officers were in the habit of testing any doubtful spot, which might be found in a case where the diagnosis was uncertain, by noting its character, duration, and whether it vesicated or not; if it did so, this was deemed, as far as it went of course, as negativing the diagnosis of typhoid fever. Two exceptional cases occurring within his own observation led him, however, to modify that opinion.

The presence of the rash seemed to import little, if anything, for it occurred in the mild as frequently as in the severe

forms of the disorder; and the cases attended with a copious rash were not at all more severe than others.

Under his remarks upon the pyrexial character of the symptoms he alludes to cases simulating a masked intermittent fever.

Headache and delirium do not appear to co-exist, nor was the latter an early symptom in any, except the worst cases, where the amount of urinary excretion was very markedly diminished. As the headache passed off, delirium—if it appeared at all—set in. Sleeplessness, dreams, and a vague sense of headache were among the earliest symptoms.

Jaundice—which is very rare in typhoid fever—was present in three cases, and in one of these, the remittent type of the symptoms during life led to the impression that the patient was suffering from a form of "bilious remittent fever."

In reviewing the symptoms referable to the digestive system he remarks, that tolerance of pressure of the abdomen was out of all proportion to the amount of anatomical lesion present in most of the fatal cases.

Omitting his observations upon the diarrhœa, the characters of the stools, and the symptoms referable to the abdomen of the typhoid fever patient, we pass to—

Hemorrhage from the bowels. Of five cases in which this was observed, four died. Great differences of opinion seem to exist as to the importance of this symptom. Inasmuch as it may arise from different sources and causes, so must the gravity of its import vary. As it exudes from an abraded fungous granulating substance—from arterial or venous implication in the ulcerating or sloughing process—so will the occurrence of hemorrhage vary in amount and danger.

Intestinal hemorrhages, from insidious ulceration, are not uncommon during apparent convalescence, and serve to indicate the great care required in diet during such times.

Under the head of *renal functions* we find nothing new. He says—the worst cases,—of an adynamic type,—cases apparently labouring under the directly prostrating effects of the disordered blood upon the nerve centres—excreted urine remarkable for the diminished amount of water and solids. In one case, which was complicated with icterus, the patient excreted only 7 oz. of urine (specific gravity 1030) in forty-eight hours, and this was albuminous, and loaded with bile pigment. He died apparently of coma, the result of uræmia. In no case of pulmonary disease was there an entire absence of chlorides. Patients (during the later stages particularly) complained of urethral scalding and difficulty in voiding their urine;

but retention of the urine, from functional paralysis of the bladder, only occurred once.

Dr. M., in speaking of the occasional occurrence of dropsy of one or other of the lower extremities, remarks, that it probably depends upon one of two causes, viz., the entrance of something into the veins from the intestinal ulcers, giving rise to embolism, or upon a spontaneous coagulation of the blood within a vein.

In his remarks upon the duration, and varieties, in point of fatality, of this fever, he says:—The cases, that were at all severe, rarely improved under three weeks or thirty days. There is great difficulty in arriving at an accurate date as to the termination of the fever. The result of clinical and post-mortem observation would lead to the inference that the typhous material commences to soften about the end of the second week, and that the ulceration and elimination of this deposit proceeds energetically, and, as it were, critically during the third week. In moderately severe cases by about the thirtieth day, the typhous material is eliminated; but the ulcers remain soft, denuded, and in various states as to vascularity for a much longer period. If the ulcerating process set up in the intestine continue, as it often does unfortunately, this is, of itself, sufficient to cause the phenomena of the fever and, in this way, a case may be prolonged until the sixth week or more, and terminate ultimately by perforation of the bowel.

In the diagnosis of typhoid fever he conceives that the chief difficulties will arise from the absence of the exanthem, or from the limited period of our observation of the patient. The first (exanthem) renders the diagnosis certain; but in the absence, even, of this we may generally arrive at an accurate conclusion by attention to the symptoms. The two diseases most resembling typhoid fever are acute tuberculosis and certain cases of pyæmia.

Dr. M. then enters into the consideration of the pathology and morbid anatomy of the disease. His observations are in accordance with the descriptions usually given.

He arranges the condition of the intestinal and mesenteric glands under two groups.

1. A deposit of typhous material in the glands, variable, but large in amount, with great local congestion or active inflammation of the mucous membrane in their neighbourhood.

2. A minor amount of deposit and swelling of the glands with, relatively, little or no inflammatory action.

When the deposit is very large, it can be seen to slough away; when less in amount, but still large, portions of the new and germinating formation, remotely placed for vascular supply, will

die in small pieces; while another portion will undergo a still finer and molecular necrosis, and the remainder, degenerating, may be absorbed or pass off with the discharge from the ulcer. So we pass to a condition in which there is a minimum amount of this morbid deposit. Judging from microscopical examination of this, it would appear to undergo a process, which Virchow terms "tuberculization," becoming a fatty, granular, and pigmental mass, and, as such, to be absorbed either without any, or with a very trifling loss of substance in the mucous membrane.

He thinks that the mildness of the general symptoms and shortness of the course of the fever, in certain cases, render it highly probable that the disorder is occasionally aborted, the amount of deposit being small enough to be removed by absorption. The fact that the tumefaction and deposit in the mesenteric glands must be so removed is an evidence that a similar process may occur in the intestinal glands.

While the different morbid states of different intestinal glands, in regard to the active local processes going on in one or more of them, may be a cause of the fluctuations observed in the patient's febrile condition from day to day, he does not think that this can be the sole cause, because such changes were observed in a few cases that proved fatal before any softening or ulceration of the deposit had taken place.

He conceives that it is to the admixture of certain products from degenerating and ulcerating typhous material with the blood, that many of the inconstant and irregular pyrexial states and local complications, occurring at a late stage of the disorder, may be due.

Of seven instances of peritonitis from perforation of the gut, he mentions one in which the perforation of bowel took place from without by the opening of an abscess in a mesenteric gland into the interior of the intestine.

Passing over his observations upon the morbid changes in the spleen, lungs, &c., we notice that he mentions three instances in which he observed the products of inflammation within the pericardium.

Under the head of air in the venous system, he describes three cases in which this existed. In all three the death was rather sudden.

The gas was not the product of post-mortem decomposition in the above cases. There were certain conditions common to them all, viz., the blood was dark and fluid throughout the body, and bubbled out from the divided veins. In the first two cases, there was an emphysematous state of the tissues of the

neck and chest, besides air in the right side of the heart, hepatic and portal venous systems, venæ cavæ, &c. The lungs were emphysematous, but no air was expelled by inflating them. In the third case, the air was only present in the abdominal veins (portal system). In this instance, the patient had lost a large amount of blood by stool and, although the lesion of the vein was not discovered, it is probable that it may have entered a vessel, through an ulcer in the bowel.

There were not any ulcers in the larynx in the above cases. Dr. G. O. Cless, in his memoir upon this subject, relates similar instances, all cases of typhoid fever, and is of opinion that the air is generated during life by a spontaneous decomposition of some of the constituents of the blood. Dr. Wilks has shewn that emphysema may result from the air entering through ulcers in the larynx. During the Crimean war, it will be remembered, that similar facts were recorded by different observers.

The remainder of this division of the paper is taken up with a description of cases which died during apparent convalescence, or were the subjects of a relapse of the fever with the exanthem.

The writer conceives that atrophy of the coats of the intestine and very grave impairment of the nutrition of the body may succeed this fever in some cases, of which he relates instances.

[Our want of space prevents our entering into the remaining portions of the paper.]

Appendix Referred to at Page 3.

Mr. Lawson—after giving a table of millesimal ratios of deaths from intermittent, remittent, yellow, and continued fevers among our troops during each year, from 1817 to 1836, at all the foreign stations—furnishes us with tables of the admissions from continued fevers at the Fever Hospitals of London, Cork, and Dublin, and the Royal Infirmaries of Glasgow and Edinburgh.

He proceeds to show the striking evidence of the passage of the pandemic wave derived from the outbreaks of disease in various civil populations.

"Thus petechial typhus was rife at Naples in 1817, and a disease denominated malignant typhus prevailed at Algiers the same year. In 1818–19 plague ravaged Morocco and the northern coast of Africa, and in 1819–20 it prevailed at Constantinople. Yellow fever prevailed in the south of Spain with

varying intensity from 1819 to 1821. There was great mortality from typhus in Ireland in 1817–18–19, and again in 1822. Fever was also very common both in England and Scotland during the former period."

"The pandemic wave, which was still sensible in the Ionian Islands and Canada in 1817, overspread the British Isles in 1817–18–19; and it will be observed that London and Cork were, comparatively speaking, affected to a greater extent in 1817 than Dublin, Glasgow, or Edinburgh; all except London had their greatest sickness in 1818, but the Scotch stations carried more of this into the following year than the Irish. The wave described previously as having passed over the Mediterranean in 1818–19 was experienced much more slightly; it seems to have kept up the admissions in London during 1819–20; in Dublin, in 1820–21; and in Edinburgh till 1822. The wave which was at the Ionian Islands in 1821 was sensible in London and Cork in 1822, and in Dublin and Glasgow in 1823. This was followed by that which was at the Ionian Islands in 1823, so closely, that fevers increased in numbers for several years, as will be shown below."

Again, between 1823 and 1829, there was a series of pandemic waves, first apparent at the Cape, from which they passed on to the northward in succession with remarkable regularity.

"In 1824 the first Burmese war commenced, and troops were sent to various points of that territory, who suffered much from fever in this and the following year. Those at Rangoon were affected early in 1824 with the 'Duegur,' or what has been called the 'Dandy fever.' This form of the disease appeared at Calcutta at the same time; the following year it appeared at Burhampore and places in the vicinity. Plague broke out at Cairo in 1824, and carried off 30,000 persons, the following year it prevailed in Lower Egypt. It was rife at Constantinople in 1826, and in 1827 reached Odessa. In 1828-29 plague was very common in Egypt, Syria, Greece, and Turkey in Europe, and affected the Russian troops in the Danubian Provinces, and in the neighbourhood of Adrianople extensively. Petechial fever prevailed at Naples in 1828. In 1827 remittents of a very aggravated type were common in the fenny districts in the south of England. In Scotland, fever was common in several of these years."

"It was stated above that the wave embracing the Ionian Islands in 1821 was sensible at London and Cork in 1822, and at Dublin and Glasgow in 1823, and that this was followed by that found at the Ionian Islands in 1823–4, so closely that sick-

ness increased for several years, attaining its maximum in London and Ireland in 1826–27, but in Scotland, as before, a year later, when the stations to the southward were again healthier. In Iceland, typhus, with some other forms of epidemic disease, prevailed in 1828–29, a year later again than in Scotland. Remittents, equalling those of the tropics in violence and unmanageableness, were common in the marshy districts in the south of England in 1827."

THE NERVES OF THE ORGANIC MUSCLES.

By Dr. Klebs, Assistant at the Pathological Institution at Berlin.

The bladder of the frog has served exclusively as the object of my researches. As the lateral parts of the bladder and the ligaments contain the larger nerves and vessels, this organ appeared particularly adapted for the discussion of the question, "In what manner are the nerves distributed to the tissue of the organic muscular fibres?"

The more important results of these researches may be stated as follows:—

1. The nerves approach the hindmost part of the frog's bladder from both sides of the rectum in several branches, anastomosing amongst each other. A main branch rising to the top of the bladder is perceptible on both sides. From this plexus, which does not extend over the foremost part of the bladder, run in all directions, a great number of smaller branches, at right angles to the ascending fibres, which divide again and again, until they only consist of one or two fibres. This formation of the branches only takes place in a very limited space, hardly extending over the third part of the posterior surface of the bladder in its full height, and growing narrower towards the top. It is but in the same limits that ganglion cells are to be found. The larger the branches of nerves in which they are included, the greater is the number of cells forming one ganglion. On the outlines of that place only a few single cellules are perceptible, either attached to one of the smaller branches, or connected with a single nerve fibre, "final cells" (enständige zellen) according to Jacubowitsch.

2. The larger branches consist throughout of double contoured fibres, the ganglion cells being precisely similar to those of the sympathetic. The connexion existing between the two can only be ascertained on the peripheral branches of the nerves.

3. The nerve fibres of the larger branches are enclosed in a tubular neurilemma, showing, under the microscope, on both sides of the fibre two lines, between which longitudinal nuclei will be perceived. The marrow-like (myeloid) substance of the fibres is separated from the neurilemma by a small transparent space, and the former in its fresh state is bounded by two stripes of a bright hue, in the middle part of which the nerve fibre assumes a transparent appearance. Counting the neurilemma as a part of the fibre, one may call it "an eight-lined one."

In the further progress of the nerve fibre, the neurilemma is getting narrower, only one line is perceptible, and the nuclei when seen from the side view are generally situated so that the pointed ends seem to extend into the visible contour of the neurilemma. On still advancing, the neurilemma joins the nerve-substance, the transparent space ceasing, and the nuclei seeming to be laid on the marrow-like substance. Meanwhile, also, the nerve-fibre has altered its qualities; only very little narrowed, it appears throughout of a plain grey colour, the bright stripes disappear more and more. Then generally follows a portion, in which the nerve-fibre affords the view of fine longitudinal stripes, an alteration which takes place rather suddenly, particularly at the division of the fibres. These latter fibres (or bundles of fine fibres) dissolve by detaching small pale fibres, which equally carrying nuclei anastomose amongst each other, and form a wide-meshed network ("net-fibres, intermediate network") showing in the knot-points for the most part a triangular accumulation of a fine granulated mass, which, however, must not be considered as ganglion-cells, because nuclei are not to be found at all places. The network of the nerves appears to be completely closed, in which formation, participate all the nerve-fibres leading to the bladder. Within that network sometimes larger accumulations of fibres make their appearance, from which net-forming fibres branch off in all directions. These masses of fibres seem to possess a different development in each individual case. In some instances I did not observe them at all. Dr. Beale, in his last publication on that subject, gives a sketch of them developed upon an extensive scale (Archiv. of Med. vol. iii, Pl. xv.).

The fibres included in these masses, are much finer than those which form the network; I estimate them at about 0,0001 mm., whilst the net-fibres are of a size far as 0,0003 mm. They are principally corresponding with the fibres of the long-striped nerves above alluded to, and this leads me to believe, that all the pale nervous fibres lying between the two consist of bundles of the

finest fibres. From the same masses sometimes radiate several fibres enveloped in the same neurilemma, which are separated by wide intervals. The nuclei were placed partly in the neurilemma, partly in the single fibres themselves. In the latter place they appeared a little more pointed, I cannot assert whether or not these compound fibres, which are similar to those described by Remak and Haerkel in the abdominal marrow of the Astacus fluviat., become transformed into pale net-fibres, by the conjunction of their fine fibres, and increasing narrowness of the neurilemma.

4. From the network-fibres the muscle-nerves branch off; some of them originate from the striped, but never from the double-lined fibres Single fibres may be seen parting from the network-fibres in a straight direction, and partly of considerable length, which never enter into contact with each other. They are going to a bundle of muscle-fibres, entering therein under a right or a little more acute angle. Shortly before entering, they show constantly a swelling of a particular kind, which distinguishes itself remarkably from the nuclei by a bright, reflecting appearance. Their form, also, is very characteristic, showing the peripheral end finely pointed, the central one, however circular, often swollen to a sort of ball. In several instances I observed in them reflecting masses, which I may call nervous knots (Nervenknoten), a round spot of a somewhat darker colour with a distinct border-line. The length of these knots exceeds that of the nuclei, and was in one case 0,0083 mm.; the greatest width was 0,0017 mm.; the thickness of the nervous-fibre 0,0003 mm.

The fibre coming out of the knot enters immediately into the bundle of muscle-fibres, in most cases at right-angles to it; divides then into two fibres, separating towards the muscle-fibres, in opposite directions. These latter, "intermuscular fibres," now form long-meshed nets, by splitting themselves dichotomously in such a manner, that the branches, parting at the place of their division, more or less rectangular, turn round again into the longitudinal direction. Each mesh of the net so formed is stretched out in the direction of the muscular fibres, but very contracted in the opposite one. The smallest meshes correspond in their width with that of one muscle-fibre, whereas they never reach their length. All nerve-fibres are going on in a wavy line, and thereby, and by their brighter appearance, they may be distinguished from the pale outlines of the muscular-fibres. The more successfully a preparation is made, the more closed will the nets be seen, whilst in preparations of an inferior kind, many of the fine nervous fibres become varicose, or even disap-

pear, dissolving themselves at last into a line of separated points. I am, therefore of the opinion, that an immediate conjunction between the nervous and the muscular substance does not exist. In the same manner, there is in the voluntary muscles a distinct partition between the two substances, notwithstanding they are in a close contact, a circumstance which may have led to that erroneous supposition, of there being a separating membrane between the two. (W. Krause.)

5. The ganglion-cells of the bladder are, as it is said above, of the same constitution as those of the sympathetic ganglia; somewhat extended cellules with a little heap of red-coloured matter; only in the peripheral parts are occasionally perceptible cells, of a smaller size, and without coloured matter. To each cell runs one double-lined or pale fibre, and from each cell parts also one of the same kind or a small number (three or four) of net-fibres (the latter are the so-called "final cells"). Therefore we have bi- and multipolar cells, but no uni-, and apolar, which is also in accordance with the observations of Dr. Beale (on the structure and formation of the so-called apolar, unipolar, and bipolar nerve cells of the frog. Phil. Trans., May, 1863; Phil. Trans., June, 1862, p. 895; Archives of Medicine, 1862).

6. The nervous fibres exhibit another relation besides that to the muscular fibres and the ganglion cells, namely to the corpuscles of the connective tissue (Bendegewebskörperchen.) To the latter go likewise small fibres which spring out from the neurilemma, which appear very often varicose; but I do not consider this particular quality as a sufficient criterion for their nervous nature, because the same quality may be found in all the fibres diverging from the corpuscles of the connective tissue, and what speaks the more against it is, that in the double-lined nerves, which have a transparent interval between the long-striped or marrow-like ventral mass, no alteration in the inner border-line could be perceived in such places. However, I by no means deny the existence of a connexion between the two structures, which, when judging from the results of physiological experiments, seems rather probable. The only decisive criterion for nervous substance is in my opinion its negative effect as to the longitudinal axis against the colours of plates of gypsum in polarised light, which is by far stronger than the positive one of the diodiaklasts of the muscle-fibre. I reserve a more particular report on this matter, and may here only mention, that this effect may be pursued much further in the peripherical extension of the nerves, than the so-called marrow-like appearance. So all the fibres which form the intermediate net, or

lead to the muscles with their respective knots, will show these particular qualities, whilst hitherto I did not succeed to prove the same state of things on the smaller intermuscular fibres.

I therefore distinguish in the nervous system of the frog's bladder, in the first place a nervous plexus, containing ganglia; in the second one, an intermediate set of nerves, parting from the first, the fibres of it probably representing bundles of fibres; and in the third place, a real intermuscular, probably terminal net, which originates from particular fibres of the intermediate net, furnished with nervous knots.

[Although the author requested me to modify his expressions to suit our language, I have only thought it necessary to alter a very few words, as I felt that by doing more, I should probably impair the author's meaning, which is very clearly expressed. It will be seen, that in the main points Dr. Klebs agrees with me, and especially as to the existence of the network, and the complete absence of any ends or terminal extremities to the nerves. He appears not to have observed the fine nerve-fibres running in the sheath of the nerves, although he has noticed what is generally regarded as the outline of the sheath on one side only of the nerve-fibre (see my Fig. 31, Plate xliv, Phil. Trans. 1862, repeated in Plate xiii, fig. 1, under *d*, in this Journal). Dr. Klebs thinks that the nerve fibres may be continuous with the connective tissue corpuscles. The results of my own observations are strongly opposed to such a view. I feel confident that there is no such connection. Dr. Ciaccio has also expressed himself against such a doctrine (see his paper on the nerves of the cornea. Trans. Mic. Society, July, 1863).—Editor.]

PATHOLOGICAL OBSERVATIONS IN A CASE OF EPILEPSY ACCOMPANIED BY DIABETES.

By J. Lockhart Clarke, F.R.S., &c.

Plate X.

This case occurred in the practice of Dr. Brown Séquard, at the Hospital for the Epileptic and Paralysed. The following particulars relating to the patient's history were supplied to me by Dr. Hughlings Jackson, assistant-physician to the hospital:—

"George M., aged 53, was admitted for epileptiform seizures, April 2, 1862. He was a well-built man, and had had

good general health until his first seizure in October, 1859. Yet he had been an intemperate man, and had had slight symptoms of delirium-tremens, but no distinct attack. There was no hereditary tendency to any particular disease. The only circumstance likely to have had an influence on his illness was that he had had two blows on the head, respectively twelve and five months before the first attack, but they were not attended by any apparent immediate effects.

"As stated, the first attack was in October, 1859. He then had 'fits' for a whole day, and for one year afterwards, he had about one daily. Then he had them oftener, two or three a day. On October 2, 1861, he had fits for the whole day, and from this time he had the fits more frequently than ever, five or six a day, and as many in the night.

"In the severe fits, viz., October, 1859 and October, 1861, he was insensible, but did not bite his tongue. They would begin by a sensation 'in the chest below the left breast, and he was in terrible pain in the stomach.' He was, it was said, first red and then deadly pale, but he did not fall, and was not insensible. He had also attacks in which his limbs were jerked out.

"Careful inquiries were made as to paralysis and defects of the spinal senses, but the only abnormal condition of the kind was that he had incontinence of urine. Of this he passed four or five quarts daily. He drank a great deal and was very ravenous for food. On July 3, his urine was examined for sugar. It was then of the specific gravity of 1037, and contained a good deal of sugar. It was frequently examined and always with pretty much the same result.

"He was subject to headache, and his temper was soured. His mind and memory generally were impaired. He slept very badly indeed.

"He continued much in the same condition until October. On October 13, I saw him at home in consultation with Mr. Powell, of Amwell-street. He had then retention of urine. When I saw him he had been relieved of the symptom, but seemed oppressed and miserable. He still had no paralysis. He had, however, had 'fits' of a different kind. 'He was shaken all over' every two minutes, for an hour. It was not shivering, but we could get no clear account of what took place; indeed, although repeated investigations were made, his wife's statements were very loose, unconnected, and vague. Next day he was incoherent and asked for his mother, who had been dead twenty years.

"On October 29, his fits, according to his wife's descrip-

tion, were as follows:—'His heart first beats violently and he shakes the bed. He turns first red and then white. He is not insensible. They occur about every quarter of an hour.'

"He continued in the same general condition, but towards the end his sight failed, and the left third nerve became impaired, but never completely paralysed. He was now so dull and stupid that it was with difficulty that I could make an ophthalmoscopic examination. I found the optic disc very much reddened, and its margin obscure. In fact the disc was lost in a reddened patch about thrice its size. There were white patches on it in some places. The arteries were very small and the veins large.

"He died April 5th, no other paralysis having been observed. It is certain that, unless at the very last, there could have have been no marked paralysis; but it is possible that a little impairment may have escaped attention, as he was for the last few weeks constantly in bed.

"After his death, I learned that he had had increased sexual desire, and this explained to me curious little scenes which occurred in my presence betwixt him and his wife. He was then so stupid that he did not seem to feel my presence, unless actually addressed."

The above history of the case was furnished me by Dr. Hughlings Jackson, after I had examined the parts which I received for investigation. These parts consisted of the whole of the m e d u l l a o b l o n g a t a and p o n s v a r o l i i, with the crura cerebri and portions of the o p t i c t h a l a m i. The l e f t optic thalamus was almost wholly cut away; but of that on the r i g h t side there remained about the two inner thirds. The right optic thalamus was softer than natural, and its softness was peculiar; for on pressure it returned a certain elastic, or kind of g e l a t i n o u s resistance. On making with a sharp razor a succession of thin sections from without inwards, I found, near the middle of its inner third, a rust-coloured mass about the size of a hazel-nut. This mass, however, was irregular in outline, and extended, in the form of a kind of tapering process, as far as the posterior border of the thalamus, where it joins the crus cerebri. On examining, under a sufficiently-high magnifying-power, different parts of this substance, in a perfectly fresh state, I found them to consist of the following elements:—1. A few partially-disintegrated, or otherwise altered, blood-corpuscles. 2. Hematoidin or colouring matter, resulting from the disintegration of blood-corpuscles. 3. Fatty substance in abundance, and in different forms. 4. Granular corpuscles, known as exudation, or the so-called compound in-

flammation, corpuscles, in considerable numbers. The hematoidin occurred in three different forms. 1. Crystallised in the form of oblique rhombic prisms of different sizes, and either separate, as at *a* (Fig. 1, Pl. X.), or embedded in masses of fat globules, as at *b*. 2. In the form of amorphous granules interspersed through clusters of oil-globules of different sizes, as at *c, d, e, f, g*. 3. In a state of solution in the substance of the oil-globules, staining them with yellow or brown of different shades, as at *b, f, g, h*.

The fatty substance, also, assumed two different forms. 1. Scattered molecules, and globules of different sizes grouped into clusters containing molecular or crystalline hematoidin, by which they were more or less tinted, as at *b, c, d, e, f, g, h, i*. 2. Acicular and radiating crystals of margarin, as at *k*. Granular or so-called exudation corpuscles are represented at *l*.

The rust-coloured mass composed of these elements appears to have resulted either from idiopathic inflammation alone, affecting the part, or from extravasation of blood, followed by inflammation. I am disposed rather to adopt the former explanation. The remaining portions of the thalamus were found to be thickly interspersed with small oil-globules, to which the gelatinous or elastic kind of resistance, already mentioned, must have been due.

The central parts of the pons varolii were somewhat softer than usual, but contained neither fat-substance nor any other kind of morbid product. A similar degree of softening extended down the central parts of the medulla oblongata, involving the middle of the raphè and those portions of the medulla on each side, which are traversed by the roots of the lingual nerves, together with the transverse commissure of the olivary bodies, lying behind the anterior pyramids. The olivary bodies themselves, however, were unaffected.

In the neighbourhood of the calamus scriptorius of the fourth ventricle the posterior parts of the medulla were considerably affected. Fig. 2 represents a transverse section of some of these parts on each side of the median line a little above the point of the calamus scriptorius. *f, e, f'*, is the median portion of the floor of the fourth ventricle; *a* is the posterior pyramid of the left side, forming the outer part of the floor. *b, b'* the vesicular nucleus of the uppermost roots of the spinal-accessory nerve of the same side; immediately below *b'* is a dark oval mass consisting of bundles of longitudinal fibres. The posterior surface of this nucleus with that of its fellow of the opposite side, here forms the point of the calamus scriptorius, and the floor of the fourth ventricle, *f, e, f'*, on

each side of the medial line. *c* is the space surrounding the elongated canal. *d* is the vesicular nucleus or centre of origin of the lingual nerve of the left side; on the opposite side its large and variously-shaped cells are represented. *g g'* are the roots of the lingual nerves. *h* is the raphè.

Now at this level of the medulla oblongata, the surfaces of the spinal-accessory nuclei *f*, *f'*, forming the median floor of the fourth ventricle, were covered with a layer of oil-globules, resulting evidently from degeneration of their superficial portions, which were ragged, uneven, and, as it were, eroded. In the median furrow also their natural substance was completely destroyed, and replaced by a deep column of densely-crowded oil-globules, *e*, which were continuous with those on each side *f*, *f*. The point of the calamus scriptorius was composed entirely of this fatty column; and a similar degeneration, but in a less degree, extended to the corresponding parts a little below, as well as a little above, where it partly affected the nuclei of the pneumogastric nerves.* All the remaining, deeper portions of the spinal-accessory nuclei *b*, *b'*, were scattered at intervals with isolated oil-globules of considerable size; and the space between them, *c*, surrounding the canal, contained them in still greater numbers. The posterior pyramids *a*, *a*, were likewise interspersed with the same kind of globules, which were to be found, also, more sparingly scattered even through the restiform bodies. The cells of the spinal-accessory nucleus were normal, many of them being filled, as usual, with dark-brown, or nearly black pigment granules.† The lingual nuclei contained no oil-globules, and their cells were normal in shape and size, but enclosed rather more pigment than usual.

NEW OBSERVATIONS UPON THE MOVEMENTS OF THE LIVING OR GERMINAL MATTER OF THE TISSUES OF MAN AND THE HIGHER ANIMALS.

By Lionel S. Beale, M.B., F.R.S.

Preliminary Communication.

The movements of the matter which I have distinguished as living or germinal matter of many of the lower ani-

* It must be recollected that the nucleus of the pneumogastric nerve is the upward continuation of the spinal-accessory nucleus from about the *calamus scriptorius*. (See my "Researches on the Medulla Oblongata," Phil Trans. 1858.)

† Without knowing that a number of dark pigmentary cells are always found in the spinal-accessory nucleus in a state of health, these bodies might be mistaken for the products of disease. It is a curious fact that I have not found them in the corresponding nuclei of mammalia. (See my Researches, loc. cit.)

mals and vegetables, are well known. I have endeavoured to show that the power of movement resides in the living particles themselves, and have expressed the opinion that these movements cannot be explained by physics or chemistry. Particles far too minute for us to see by the highest powers to be obtained (5,000 diameters), must possess the inherent moving power, the effects of which we observe in the changes occurring in the form of the masses of living matter which are but the aggregate of such minute living particles. It seems to me probable that the most minute living particle which it is possible to conceive, is a spherule, and that this spherule is capable of altering its form. I believe that the alteration results from the influence of wonderful inherent internal powers, of the nature of which we know nothing as yet, which wonderful powers may at least, for the present, be termed "vital," to distinguish them from physical and chemical properties.

I am now in a position to extend this view to the germinal matter of tissues.

I have already stated, that living matter possesses the power of altering its form, and movements have been observed by Brücke and many others in white blood, chyle, and lymph, corpuscles. They can be seen easily in pus and mucus corpuscles, young epithelial cells,* the nuclei of the corneal tissue as shown by Kühne, and indeed the germinal matter of most of the tissues of the higher animals, as shown by the difference of form exhibited under different circumstances, and the existence of protuberances, buds, or offsets. Every one is familiar with these movements in the lower forms of life, but the general conclusion I have arrived at, that the matter which is the seat of these movements is the living or germinal matter, as contradistinguished from the lifeless formed material, is not yet generally admitted.

I have already described, with some care, the movements occurring in the germinal or living matter of the mucus corpuscle from the human throat, and have stated that I had "seen an entire corpuscle move onwards in one definite direction for a distance equal to its own diameter or more."—(British Medical Journal, March 28th, 1863.) Often, as the mass of germinal matter moves, it leaves a sort of thread behind it. This thread I consider to be the lifeless formed matter which results from the death of a portion of this living, moving, growing mass under, certain conditions.

Now just as this living matter of the mucus corpuscle moves

* See my paper in the British Med. Journ., March 28th, 1863.

away, leaving its formed material behind it, so the white blood corpuscle may move and leave fibrin behind it, and the germinal matter of fibrous tissue may leave, as it moves in a definite direction, its proper formed material, which gradually undergoes condensation into fibrous tissue.*

I have evidence to prove that the formed material of many other tissues is produced in precisely the same way. I shall shortly publish the results of observations which establish this most important fact in fibrous tissues generally, in lymph, in the fibrinous clot, and in the formation of connective tissue, in muscle, in all nerves and nervous centres (see also my paper on "The structure and formation of the so-called apolar, unipolar, and bipolar nerve cells of the Frog," Phil. Trans., 1863†), in the formation of dentine and in many other tissues; so that I feel convinced the fact is a general one, and that I shall have no difficulty in proving it by demonstrating its results in the tissues of any living being.‡

That my meaning may not be misunderstood, I will particularly adduce the case of striped muscle and elastic tissue. Kölliker maintains that in the formation of the latter tissue nuclei take no direct part. I can affirm that what are called nuclei take the same part in the formation of this as in the case of other tissues. I can very clearly demonstrate the nuclei in the ligamentum nuchæ of the lamb, in specimens which have been immersed in carmine.

The mass of germinal matter of muscle seen in Fig. 1, which occupies the interval between *b* and *c*, has moved there, for

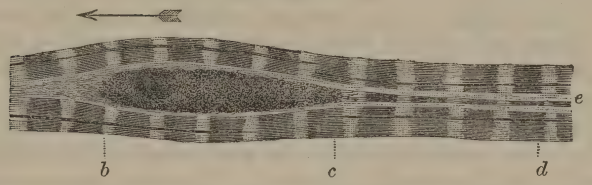
* The two masses resulting from the division of a mass of germinal matter move in opposite directions; and thus in a mass of tissue, when movement can occur only in opposite directions, the oval masses of germinal matter will alternate with each other throughout the texture; one end of the mass is often blunt and rounded, while the other gradually tapers into the thread of formed material.

† This memoir will shortly be published in a separate form.

‡ In many secreting cells, as, for example, in the long mucus secreting cells upon the fauces of many of the reptiles (they are very large in the boa), the so-called nucleus, or mass of germinal matter, is near the lower or attached portion of the cell; while, extending towards the surface, is the long wide tube of the cell, often open at its free end, which contains the product of secretion. In this case the pabulum is taken up from below, and absorbed by the deep surface of the small mass of germinal matter. At the opposite surface the germinal matter undergoes conversion into formed material, which pushes that which was produced before it onwards towards the free extremity of the cell. The germinal matter remains stationary, but movement in a definite direction is given to particles resulting from its death. Ciliary movement is probably a phenomenon very closely allied to the above; and, I believe, in the absorption of matters by the cells of the villi a similar change occurs, but, of course, in the opposite direction. The position of the mass of germinal matter (nucleus), with respect to one or other extremity of the cell, will depend upon the position of the pabulum. The nucleus is usually nearest that extremity of the cell which absorbs the pabulum.

it was once in the space between *c* and *d*. As it moved, part has undergone conversion into the contractile formed material *e*, and a continuation of the contractile material would, if the muscle had lived, soon have occupied the position between *b* and *c*, while the mass of germinal matter would have moved still further on. At the same time that it advances it absorbs nutrient matter, so that much formed material or contractile tissue may be produced, without the germinal matter altering in size.

Fig. 1.



Portion of voluntary muscle, from the midst of an elementary fibre of the frog, very highly magnified, showing the manner in which the contractile tissue (fibrillæ) is formed from the germinal matter. Mass of germinal matter occupying the space between *b* and *c*. It has moved to its present position, and once occupied the interval between *c* and *d*. *e*. Contractile tissue, which was recently in the state of germinal matter. Over *c*, the conversion of germinal matter into formed material (contractile tissue) is actually proceeding. The mass of germinal matter is moving in the direction indicated by the arrow.

Fig. 2 is a portion of an elastic fibre of the ligamentum nuchæ, with its mass of germinal matter *a*, which is moving in the direction of the arrow, while part gradually undergoes conversion into the elastic material of the fibre which in this way becomes thickened.

Fig. 2.

a

Portion of a fibre of yellow elastic tissue from the ligamentum nuchæ of the lamb. The mass of germinal matter *a* is moving in the direction of the arrow. At the posterior part the formation of yellow elastic tissue is proceeding; the fibre, increasing in thickness as new formed material results.

Similar phenomena occur in a clot in which fibrous tissue is to be formed. Masses of the germinal matter, probably descended from the white blood corpuscles, move in segments of circles and form fibres of connective tissue.

The details upon which I rely for establishing this most

important fact, in connection with the formation of the tissues, are most interesting, and will, I think, especially when considered in connection with many phenomena familiar to all observers, be admitted to be very strongly in favour of the general conclusions I have been led to adopt.

ANATOMICAL AND PHYSIOLOGICAL CONTROVERSIES.

It is doubtful if at any previous period so many incompatible views were entertained upon questions of paramount scientific interest and importance, as at the present time. Nor are the differences which exist slight or unimportant, affecting mere matters of detail. Great principles, which can be very clearly enunciated in few words, are involved, and many of these influence the very foundations of physiology. Careful observers are at variance upon matters of observation, and many questions of the utmost importance to physiology and medicine are in the most unsatisfactory state. Rival observers originate, and rival pupils support, opposite doctrines; warm and exciting conflicts disturb the steady quiet necessary for true advance, and continual working in circles, retards real scientific progress. In no branch of science is this very unsatisfactory state of things more apparent, than in minute anatomy, and as a necessary consequence, grave doubts are cast upon the value of direct anatomical observations as a means of discovering truth, and the great differences among workers upon fundamental principles, foster, and, to some extent, justify, scepticism on the part of readers and learners.

A careful perusal of many of the articles in small print in Kölliker's Anatomy, for example, will enable any one to form some notion of the terribly conflicting views now entertained upon what we all look upon as simple anatomical questions affecting the very foundation of physiological science. If the reader will be at the pains of comparing some of these articles in the last with the corresponding ones in the first edition issued of this work, he may, perhaps, be able to form some idea of the character the work will assume ten years hence. For if observers increase in number as they have increased lately, and if, as is really not improbable, there yet remain many p o s s i b l e views upon every subject which have not yet been put forward, it will be necessary to devote an entire volume to the consideration of each separate tissue and organ. We cannot but feel that it is the duty of all who work at minute

anatomy, to make an effort to arrive at definite conclusions on wide points of general interest, and it seems to us, that many of the complex views now held might be simplified without difficulty.

It is a remark that will, perhaps, be received with some surprise, but it is nevertheless true, that conflicting views result principally from insufficient work. Observers often write upon controversial subjects after devoting a very short time to observation, and many instances could be alluded to, in which conclusions, resulting from years of thought and labour, were set aside by authorities after a most imperfect consideration of the question at issue. After a time the true view is, of course, revived, and finally accepted, but what time and labour have been for ever snatched from true progress in the meanwhile!

It is well known that Henle has recently reopened the whole question of the anatomy of the kidney. If his conclusions are correct, the views which have been taught upon the anatomy and physiology of this organ during the last twenty years, will require to be modified greatly, and not only so, but the pathology of renal diseases must be re-studied from a new point of view. Now surely there are specimens already prepared in various collections, which would confirm or refute Henle's observations. Would it not be possible for those interested in this question to send one or two specimens to some general meeting of anatomists, which specimens could be examined by the observers present? Professor Henle would doubtless send some of those upon which his inferences rest. We do not say that the question at issue might in this way be conclusively determined and completely set at rest; but, at any rate, observers would be in a far better position to judge of the correctness of these new observations than they are at present. Professor Henle is doubtless aware that such an arrangement as he supposes to exist in man, in the horse, and in the pig, cannot possibly exist in the newt, in which animal a tube can be traced in every part of its course quite uninterruptedly from the Malpighian body to its terminal portion, and injections can be forced quite through it from the Malpighian body. Surely this question of the existence of tubules without efferent ducts in the particular kidneys above named, is one which might be determined by any observer who was an adept at injection, and would devote to it a few weeks' careful work.

Even greater differences of opinion still exist upon the anatomy of the liver, and many anatomists do not admit the existence of inter-lobular ducts in all the inter-lobular fissures,

and few agree that any ducts pass into the lobules themselves. This is an anatomical question, which perhaps can only be fully elucidated in the course of a controversy between those who have studied the subject and have arrived at different conclusions.

But differences of opinion equally striking exist among living observers with regard to the minute structure of almost every organ and tissue, not only of the higher but of the lowest and simplest organisms. The physiological views deduced from anatomical observation and physiological experiment, are equally conflicting.

The connective tissue theory, the distribution of nerves, and their relation to the anatomical elements of the tissues, especially to connective tissue corpuscles and epithelium, the mode of formation of yellow elastic tissue, the mode of the distribution of nutrient pabulum to connective tissues, the manner in which nutrient matter is absorbed by the columnar cells of the villi and passes into the lacteals, are instances of great anatomical questions upon which the most conflicting and incompatible views are entertained by different observers, and taught in different schools.

Yet, with all this doubt upon elementary matters, and in spite of the difficulty of arriving at the truth upon mere questions of fact, it is astonishing, with what outrageous confidence opinions are expressed and assertions made, upon matters which real students would truly regard as the highest and most difficult problems to be solved. We have a whole army of conscientious observers utterly puzzled as to what happens when the cell of a leaf or the germ of a simple vegetable organism increases in size by growth; while, on the other hand, the most abstruse and difficult questions which ever have been, or which ever can be, presented to the mind of man to solve, are disposed of by another class of philosophers in the most off-hand manner possible, and rendered clear to the comprehension of a child, as if by magic. There is no longer any necessity for naturalists to spend their lives in studying the life-history of a few species, or for minute anatomists and physiologists to peer into the inmost recesses of living creatures. Living organisms upon this earth exhibit in the changes which take place in them nothing different to what occurs in stones, in planets, and in suns. We live in an age which to posterity will appear the most marvellous the world has ever seen. The views now put forward by most popular philosophers are destined to stand for ever unshaken. The end of all knowledge has been

reached. A new philosophy has appeared, as perfect as that which preceded it was incomplete. Our scientific successors will doubtless have reason to respect our work far more than we have to respect that of a former generation. We know how much that was believed by our predecessors has been demonstrated by us to be unsound, but those who succeed us will doubtless find that our views will stand unshaken in their beautiful and simple perfection. Those now living have seen vast problems explained, so that those who follow will have but to admire, and contemplate, and wonder at what has been done by philosophers of the present era.

There appeared to some naturalists and physiologists difficulty in explaining how Darwinian monads first became living, and although it is obvious that a few particles of organized matter coming together might produce a living creature, it was somewhat difficult to account for the formation of the organized particles before life began upon the earth. But a more sublime discovery, and a more stupendous generalization, has been made by the physicist, and henceforth biology and physiology will be obsolete; for biological phenomena henceforth belong to physics. By life we merely understand certain changes, physical, chemical, electrical, thermal, &c., which can be precisely imitated in the laboratory with precisely similar results. Vital power no longer excites the speculations of the physiologist, or the wonder of the profound metaphysician. Every tyro now knows that life is but another form of motion, and that the changes taking place in living things differ from those occurring in stones only in degree. The same mighty forces are at work in the organic and in the inorganic world. The formative power of the living organism is but a form of "heat," without which nothing living could exist. We know the conditions under which the crystal forms, and we know the circumstances under which the egg will develope.

Particles of inorganic matter arrange themselves in a certain order, and give rise to certain definite and constant forms. Nuclei arrange themselves in rows, become invested with surrounding substance, and thus a living tissue is built up! The complex tissues of the higher animals scarcely differ more from the simplest organisms than the latter from inorganic matter. In all things changes occur. In living beings these changes take place with greater rapidity, owing to the different conditions under which the matter is placed.

The real nature of the mysterious "vital forces" of a former day is now fully explained by the new discoveries of the physicist, and the true relation of these forces to the one great primary agent, simple motion, has been incontrovertibly established. In fact,

no one who is able to appreciate the marvellous results of recent physical investigation can fail to accept the inevitable conclusion, that life itself is but another form of that marvellous energy we receive from the sun. Vital action is only motion peculiarly conditioned. Like other kinds of force, it is neither created anew nor can it be annihilated. When a living thing dies, its equivalent of constructive organizing force changes its form, and either returns again to the more simple state of heat, or manifests itself in the vital actions of some lower forms of life. The living fungus growing upon the dead branch contains its equivalent of that energy which was working in the branch when it was alive. Thus it is that there is an ever-changing expression, but a never-varying quantity of force. It may be fungus life, tree life, animal life, or man life, but equal weights of fungus, of tree, of animal, and of man, will contain of course different equivalents of force.

All this energy, which is being continually supplied to our earth, and is here conditioned in so many different ways, emanates from the sun. He not only moves the steam machine made by man, but he forms and then moves the living machine. He is the chemist, who not only works in our cell laboratories, but forms them. At his bidding the cell laboratories multiply in number, and he gives to each a portion of his mighty energy. He dissolves, he evaporates, he distils, he condenses, he dries, he ignites. He arranges the retort, he applies the heat, he rises in the vapour, he falls in the distillate. By his influence, elements which were firmly linked in combination silently separate, take up their appointed positions, and assume relations which they never had known before. Some of his rays disappearing in the process, and assuming the condition of latent internal energy, new compounds result, which the synthetical powers of the most expert modern chemist have scarcely yet enabled him to produce in perfect purity; but without doubt defects in the existing chemical processes will soon be remedied, and protein compounds, now regarded as peculiar to the living organism, will be obtained in a state of purity in a crystalline form.

Nor does the sun merely play the part of cell laboratory builder, cell laboratory multiplier, and cell chemist. His work is more general and far more varied than this He *forms* the most simple and most complex structures of living beings. The petals of the rose as well as its delightful perfume, the shell of every diatom, the spicules of a hundred sponges, are his workmanship. He oscillates in the monad, he vibrates in cilia, he revolves in the rotifer. The human brain, like the forest, is

built by him, and we ourselves consist of nothing more than inorganic matter and a fraction of his energy. The formation of tissue, the action of all the different organs, their disintegration, the different phenomena observed in the same structures in health and disease, are easily accounted for by the different conditioning which the sun's force undergoes as it passes through our bodies. The soul-inspiring strains of the poet and the wisdom of the sage, the ravings of the madman, are but flickerings of rays emitted by the sun. It is very easy to understand how a minute particle of matter, so very light that many hundred times its weight would fail to turn the most delicate balance ever made, and so very minute that it is scarcely visible under any magnifying power, being supplied with a fraction of such marvellous energy, must form into a plant, or a tree, or a man. But we need not stop here, for we know how asteroids, moving with intense velocity, meet together and form suns. The immense energy which now takes the form of heat instead of motion, radiating into space and falling upon distant worlds, is conditioned in its halting-places, and becomes in its turn vegetables and animals, which afterwards melt into air, and thus give up again every fraction of force they had received.*

It is needless to pursue further the results of modern discoveries upon our notions of life. The term 'vital power' can no longer be used, for as it emanates from, and is produced by, the sun, it will be henceforth better known as sun-force. All things living from the monad to man are children of the sun. We have thought it well to give an outline of these views which have been recently rendered so clear, because the nature of vital actions has been so fully explained that it is not likely to become again a subject of controversy.

But there still remain many physiological questions of the utmost practical importance to those who have to study and treat disease, which are, as yet, undecided; and there can be no doubt that some of these might be in great measure cleared up by fair but temperate discussion. There are many simple remedies, with regard to the action of which upon the organism much yet remains to be learned. We are not yet acquainted with the action of the mineral acids and alkalis, and we all know that the very same cases would often be treated with acids by some physicians, and with alkalies by others. Then there is the all-important question of the external and internal action of alcohol in disease. What differences of opinion there are

* See Professor Tyndall's Lectures at the Royal Institution, on "Heat considered as a Mode of Motion," pp. 433, 434.

upon the action and practical value of this remedy. It ought to be possible to obtain some principle which would guide us as to the quantity that should be given in various conditions. Many practitioners have asserted that no patient, under any circumstances, should take in twenty-four hours more than a certain number of ounces. Some may think that those who enforce such dogmas perhaps rely too much upon mere opinion, since it is obvious that the weight of the individual, the activity of the processes of respiration and secretion, must affect the proportion that ought to be given. It is, therefore, clear, that the quantity which should be given must vary in different cases. Here is a practical question of the utmost importance, which can only be settled by discussion.

Temperate discussion between those who really work and earnestly think upon these great questions, will alone enable us to arrive at the truth within a reasonable period of time. Those who put forward conclusions usually support them by facts and arguments, but the real value of these facts and arguments can only be ascertained by the most careful and stringent examination of the evidence upon which they rest. In many of the questions alluded to, it is clear that at the present day two opposite opinions may, so to say, be tenable; that is, the facts in favour of each seem to be of nearly equal value; but it is clear that directly opposite conclusions upon a given question cannot both be true, and therefore new work is required. There is no method by which the direction in which new work should proceed is so soon elicited or so clearly defined as by controversy. In this way alone can opinions which are true be fully substantiated and proved to be true, and worthy of general reception. In this way alone can conclusions which are erroneous, be disproved, and demonstrated to be unworthy of confidence. By free discussion alone can hopeless working in a circle be stopped. It is, therefore, the intention of those connected with this journal to insert from time to time controversial papers of scientific or practical interest. The differences entertained will be expressed as clearly as possible, the papers will be freely illustrated, and every effort will be made to bring the points under discussion to a distinct issue.

AN ANATOMICAL CONTROVERSY.

THE DISTRIBUTION OF NERVES TO VOLUNTARY MUSCLE. DO NERVES TERMINATE IN FREE ENDS, OR DO THEY INVARIABLY FORM CIRCUITS AND NEVER END?*

By Lionel S. Beale.

By no means the least interesting of recent anatomical controversies is one which relates to the distribution of nerves to the voluntary muscle of vertebrate animals. In this particular controversy I take the warmest interest, for the subject was reopened by me in 1859 (see paper in Phil. Trans. 1860, p. 611). The conclusions I then arrived at were opposed to the views entertained by most authorities, especially Kölliker, Gerlach, and Kühne. My paper was followed by a communication from Kühne, in which he supported, by investigations upon the breast muscle of the frog, conclusions advanced by him previous to the publication of my paper, in favour of the view, that the nerves in insect muscle terminated in ends beneath the sarcolemma—in fact, were continuous with rows of nuclei which lie amongst the contractile tissue. In a note to my first paper (Phil. Trans. 1860, p. 611), I have stated that in the muscles of the blow-fly "the nerves do not penetrate into the interior of the muscle, but are connected with some very large spindle-shaped cells on its surface. These are very difficult to demonstrate, but I have been able to preserve some specimens which show this point very clearly." A month after Kühne's paper appeared, a memoir was put forward by Kölliker, in which he agreed with Kühne as to the termination of the nerves by ends, but with me as to the fine nerve fibres being upon or external to the sarcolemma, instead of penetrating through this membrane and coming in contact with the contractile tissue. Here then are three inferences upon this question of anatomical observation utterly incompatible with each other. It might be said that all are wrong; but it is an absolute necessity that two are wrong, since no two of the following arrangements can coexist:—

1. That the nerves terminate in ends external to the sarcolemma.

2. That the nerves terminate in ends beneath the sarcolemma.

3. That the nerves do not terminate in ends at all.

* The reader is recommended to study Plate XI, with the explanation, before reading the paper.

Within the last year many new memoirs have appeared, in which different, and in some cases new views are put forward; but it is undoubted that the authors all differ from me in a greater degree than they differ from one another.*

Such opposition upon a question of fact is greatly to be regretted, and it seems to me that the only way in which the question can be cleared up is by renewed investigations and increased work.

I for my part cannot yield, for the simple reason that I have seen what I have figured, and have indeed found no difficulty in following fine fibres structurally continuous with dark-bordered fibres for a long distance beyond the point where Kühne, Kölliker, and others, make them end. Moreover, my specimens show the arteries and capillaries as well as the nerves, and I have now worked at this anatomical point so long and so hard, that I have a right to ask that my opponents should prepare the specimens by the same process that I have followed, and give drawings of what they observe. I would then reply by giving drawings of the very same structure, showing what I have seen. Independent observers would then be in a position to judge between my opponents and myself.

The great question is as to the existence or absence of ends

* Kühne, Ueber die peripherischen Endorgane der motorischen Nerven.—Leipzig, 1860.

Theodor Margó, Ueber die Endigung der Nerven in der quergestreiften Muskelsubstanz.—Pest, 1862.

Kölliker, Untersuchungen über die letzten Endigungen der Nerven.—1862.

Naunyn, Ueber die angeblichen peripherischen Endorgane der motorischen Nervenfaser, in Reichert und Du Bois Reymond's Archiv. 1862, p. 481.

L. Beale, Further observations on the distribution of nerves to the elementary fibres of striped muscle.—Phil. Trans., June, 1862.

Krause, Ueber die Endigung der Muskelnerven.—Henle and Pfeufer's Zeitschrift, 1863, p. 136.

Th. W. Engelmann, Ueber die Endigungen der motorischen Nerven in den quergestreiften Muskeln der Wirbelthiere.—Centralblatt f. d. Medic. Wissensch, 1863.

L. Beale, "On the anatomy of nerve fibres and cells, and on the ultimate distribution of nerve fibres."—Quarterly Journ. of Mic. Science, April, 1863.

L. Beale, Further observations in favour of the view that nerve fibres never end in voluntary muscles.—Proceedings of the Royal Society, June 5th, 1863.

L. Beale, Remarks on the recent observations of Kühne and Kölliker upon the termination of the nerves in voluntary muscle.—Archives of Medicine, vol. iii., p. 257.

Th. Wilhelm Engelmann, Untersuchungen über den Zusammenhang von Nerve und Muskelfaser.—Leipzig, 1863.

Kühne, Ueber die Endigung der Nerven in den Muskeln.—Virchow's Archiv., Band 27.

Kühne, Die Muskelspindeln.—Virchow's Archiv. Band 28.

Kühne, Der Zusammenhang von Nerv. und Muskelfaser.—Virchow's Archiv., Band 29.

or terminal extremities. My original paper was to show that no ends were demonstrable—that from every oval "nucleus" a fibre could he traced in opposite directions, and I was compelled to infer that the fibre was endless. The appearance was that of a network. Some appear to have concluded that by the term "network" I meant that amongst the muscular fibres existed a common network, with which, at various points, dark-bordered fibres were continuous, just as small arteries and veins are connected with the capillary network at various points. This is an erroneous inference, as will be seen by reference to my first drawings. The network is like the network of lace, every thread being composed of finer threads, but in no case does a single thread pass completely round one mesh or interval. It may form part of the boundaries of many spaces, but not the entire boundary of a single space. Subsequent research in different tissues, and in many different animals, has convinced me, not only of the truth of my original conclusions respecting the distribution of nerves to muscle, but of what is far more important; and I will now take up the question in a much broader way. I assert that nerves do not terminate anywhere in free ends; and I am ready to study by my own processes of investigation any tissue of any common animal of any class that a fellow-worker may select, and discuss this large question by the publication of drawings; or if several observers will meet and agree to investigate any one particular tissue, and publish their drawings and results by a certain day, of course allowing sufficient time for investigation, my drawings and conclusions shall be in print at the appointed time. In this way such disputed points would soon be brought to a distinct issue.*

It is impossible to over-estimate the importance of this general question, for in it is involved the typical arrangement of every nervous instrument. So far from there being distinct ends I believe that in all cases "complete circuits" exist, and that in these circuits are included central nerve cells and peripheral nerve cells, generally termed nuclei, which are connected by intervening fibres. The course of any given fibre may be extremely complicated, and there may be many minor circuits connected with the greater one, but in all cases there is a circuit. A nerve never ends.

In the controversy connected with the arrangement of nerves in voluntary muscle, I think I have been somewhat incon-

* It is of course absolutely necessary that some species, easily procurable in all parts of Europe, should be chosen, and that drawings be published. The illustrations can he executed in any way the observer may desire.

siderately dealt with by some of my friends in Germany. Kühne especially has unceremoniously disposed of me by saying that the nerve fibres I described in 1860 are "vascular nerves." Now if any one will refer to my drawings (Phil. Trans., 1860), he will see the vessels as well as the nerves delineated, and if he will count the nerves and vessels, and observe the position of the nerves and vessels, and the vascular nerve distributed to a capillary in one of the figures, he will indeed be puzzled at the conclusions of my friend.*

Again, Kühne considers that I have mistaken connective tissue and its nuclei for nerve fibres and their nuclei. He sets me right by telling me that a fibre which I have seen exhibiting a very definite structure and arrangement, is nothing but indefinite connective tissue. Now I have very fully considered, in several of my papers, the relation of nerves to connective tissue, and the distinction between the finest nerve fibres and connective tissue, so that I need not minutely discuss this part of the question here. Let the reader refer to my drawings (1860), and he must conclude, either that the fibres I have delineated are not connective tissue, or that I have most wilfully misrepresented connective tissue.

Again I would venture to remark, that not one of my opponents has condescended to investigate the structure in the manner I stated was necessary to see the appearances I delineated, namely, by soaking the tissue in syrup or glycerine, so that not one of those who correct me has seen the fibres I have described. To this day the finest nerve fibres I demonstrated in the diaphragm of the mouse, have not been seen by those who have since taken up the same investigation. And why, I would ask, do not my opponents study the same muscle in the white mouse. I have tried many others, and have found that the diaphragm is most easy of investigation. If this muscle be carefully studied, such plexuses of dark-bordered and fine pale nerve fibres will be found as will astonish the observer.

I have very frequently dwelt upon the importance of injecting the capillary vessels with transparent injection; and before I commenced my first paper I had thoroughly convinced myself,

* Strange to say that at the time my friend passed these severe criticisms he had not seen my drawings in the Phil. Trans. for 1860, which however were not published until 1861—Kühne's memoir is dated 1862. In page 28, after referring to the abstract of my paper published in the Proceedings of the Royal Society, he says: "Deren Original mir leider, nicht zugänglich ist." As it is evident my original drawings in the Phil. Trans. are not known to many of those who have been writing papers upon this anatomical question in Germany, I propose shortly to republish the most important of them.

that it was not possible to make out the ultimate distribution of the nerve fibres in recent specimens examined in water, serum, vitreous humour, and the like (1859). After performing many experiments I was compelled to accept the conclusion, that such minute anatomical points could only be demonstrated in specimens immersed in syrup, glycerine, or some such viscid, highly refractive material, miscible in all proportions with water. By this means I was enabled to subject the section to very great pressure, and reduce it to the tenuity required for examination by the highest powers (more recently $\frac{1}{2}\frac{1}{5}$ and $\frac{1}{5}\frac{1}{0}$) without destroying the most delicate structure. By comparing uninjected and injected specimens of the very same tissues, I have proved that capillary vessels may be readily mistaken for fine nerve fibres, and that when shrunk and very much stretched, it is not possible to distinguish them as capillaries at all. In this state they often appear like fibres of connective tissue.

I have also proved that when the transparent injecting fluid, which contains glycerine and a little chromic acid, is introduced into the vessels immediately after death, it permeates the thin capillary walls, and preserves and hardens slightly, all the delicate tissues, including the nerves. In this way alone can a highly refracting fluid be uniformly distributed to all parts of a tissue immediately after the death of an animal. To this mode of preparation I was gradually led many years ago, after trying very numerous experiments.

Next I would respectfully submit that those who correct me will insist that the nerves stop short at a point a very short distance from the dark-bordered fibre, although I have stated over and over again that in my specimens they may be followed a long way beyond this point. In fact my correctors will make the nerves end a very short distance from the point where I conceive the really important part of a most extensive plexus system of fine nerve fibres only begins. The finest nerve fibres distributed to the muscles of the frog for example, are far finer than is supposed in Germany, and the fibres delineated by Kölliker, Kühne, Engelmann and others as single fibres, really consist of several fine fibres. Kölliker admits the existence of pale nucleated intermuscular fibres, first described by me, and agrees with me in the opinion that they lie upon the external surface of the sarcolemma. According to him the delicate sheath is prolonged upon the pale fibres, and he infers that here and there at least a thin layer of the white substance extends along the pale fibre. Kölliker very rightly distinguishes the nuclei of the pale fibres (*c*) from the nuclei of the muscle (*d*) (Plate XI, fig. 2), while Kühne had mistaken the latter

for his peculiar 'nerven-end-knospen.' But, in support of his former views, Kölliker insists that the pale nerve-fibres terminate by free extremities outside the sarcolemma.

Kölliker regards the ends he represents as real natural terminal extremities, but I think that his own drawings contradict his conclusions, for the abrupt ends represented by him are really much more like accidental breaks than true terminations. In my own observations upon the breast-muscle of the frog, the nerve-fibres are also represented as ceasing (fig. 3, Plate XI); but I feel quite sure, from observations upon other specimens, that they continued farther. I could not follow them in particular instances, in consequence of the extreme delicacy of the nerve-fibres, and the thickness and refractive power of the large muscular fibres upon which they ramified. My own specimens are figured in Plates XI, XIII, and XIV, also from the breast-muscle of the frog.

I shall endeavour to show (see page 175) that the lines which Kölliker supposes to be the outline of the sheath, result, in many instances, from the presence of very fine nerve-fibres, which form, with the fine fibres resulting from the division of the dark-bordered nerve-fibre, fine compound nerve-fibres which are distributed upon the surface of the sarcolemma as a network or plexus. Compare the nerve-fibres in Kölliker's figure copied in Plate XI, fig. 2, with my figures 2, 3, 4, Plate XIII, and figs. 7 and 9, Plate XIV.

The main difference then between the distribution of the nerves upon the elementary muscular fibres of the breast-muscle of the frog, and those of the diaphragm of the mouse, described in my paper published in the Phil. Trans. for 1860, consists, according to my observations, in the size and number of the nerve-fibres distributed to a given amount of contractile tissue. In the frog the difficulty of demonstration results from the extreme fineness of the nerve-fibres, which ramify upon the sarcolemma, and from the very few nuclei, as compared with those in connection with the nerves of the mouse. The distribution in essential points is the same, and my latest observations confirm the results obtained in my first paper in all essential particulars. The nuclei of these nerve-fibres, and the fine fibres themselves, have been considered by many German observers as belonging to the connective tissue beneath which they lie, but no one in Germany has as yet attempted to discuss this part of the question in detail, indeed, from the observations made by some, it would seem that they had neither patience nor inclination to enter upon it.

The nerve-fibre, represented by Kühne as passing through

the sarcolemma of the muscular fibre of Hydrophilus, consists of a bundle of very many fibres which lie upon the surface of the sarcolemma, and of branches of tracheæ which are not delineated in his drawings. There is no single nerve-fibre in the insect nearly so wide as delineated by Kühne, and what he terms the axis cylinder of one nerve consists in all probability of several entire nerve-fibres. The bundles of fibres delineated by him in his Plate II., figs. 9 and 10, as ending in the elementary fibre are, I think, a considerable distance from their finest terminal branches.

It is not possible to see many of the finer bundles of insect nerves in the mode of examination adopted by Kühne. In the last number of the Microscopical Journal (October, 1864) will be found a paper by me upon the structure of the sarcolemma, and the arrangement of the nerves in insect muscle, in which this question is considered.

The peculiar bodies described as 'nerven-end-knospen' are in some cases the nuclei of nerves upon the sarcolemma, in others the nuclei of the contractile tissue beneath the sarcolemma. With regard to the very peculiar structure of those represented in the drawing, in Kühne's plate, in his memoir 1862,* I can offer no opinion, as I have never seen anything at all approaching the representation.

I believe that Kühne's pale terminal fibres really consist of bundles of very fine fibres which divide and subdivide into still finer compound fibres, which are so very fine as not to be visible in specimens as usually prepared.

I might criticise many of the drawings separately, but will only offer the opinion that the dark-bordered fibres made to terminate in fig. 12, A. B. C., Plate II., are a long distance from their finest subdivisions.

Kühne has recently reiterated his conclusions in another paper (Ueber die Endigung der nerven in den Muskeln. Virchow's Archiv. Band 27), which is illustrated with several drawings. I regret that I have not been able to confirm the conclusions at which he has arrived in the memoir. Nor have I ever been able to see the nerves in fresh muscles with the distinctness given in his drawings. I have seen the numerous cells or nuclei represented near the dark-bordered fibre, as shown in his figures, but they were arranged more regularly in my specimens, and in lines, and between them were very pale granular fibres. They were on the surface of the sarcolemma, and as the vessels were injected I could demon-

* By a misprint in the note on p. 162 the date of this memoir is stated to be 1860 instead of 1862.

strate very positively which nuclei belonged to the nerves and which to the capillaries. I have stripped off from the fibre such collections of cells, leaving the sarcolemma with its contents behind. I can assure Kühne that I have traced fine pale fibres from dark-bordered fibres, not so wide as those represented in fig. 11 of his plate, over thirty or more elementary muscular fibres, in the mylo-hyoid muscle of the Hyla (see Plate XII), and that what he terms the nuclei of the nerve-sheath are, in many instances, the nuclei of fine nerve-fibres running in the sheath, which are much nearer to their terminal distribution than the dark-bordered fibres which he makes to end beneath the sarcolemma. See fig. 1 at *d*, figs. 2, 4, 6, Plate XIII.

But it is necessary for me to enter more into detail with regard to the arrangement described by Kühne, in his last two papers, for it will be noticed that in these last communications, appearances very different to those advanced in the original memoir are delineated.

From the paper in the 27th volume of Virchow's Archiv, Plate XI., I have selected certain figures, 1, 2, 3, and 4, from the rabbit, and fig. 7 from the dog. The most important parts of these figures have been copied in Plate XI., figs. 4, 5, 6, 7.

With regard to the fig. 1 copied in Plate XI., fig. 6, I am of opinion—1. That the nerve never ends abruptly in any case, as represented; 2. That the nuclei represented as beneath the sarcolemma are really upon it, that is external to it; 3. That these nuclei have been disarranged in the process of preparation.

With regard to his fig. 2 (copied in Plate XI., fig. 7), I would remark in addition, that the dark line which represents the outline of the white substance is continued as the outline of the sarcolemma, an arrangement which most certainly does not exist in nature.

In Kühne's figs. 4 and 7 (copied in Plate XI., figs. 4 and 5), the dislocation of the nuclei is clearly represented.

The 'nervenhügel' H, delineated in these drawings, are not represented in Kühne's earlier papers. In the four drawings, *a* is considered by Kühne as the nuclei of the nerve sheath, but (at least in some instances) these are nuclei of fine nerve-fibres running in the sheath, as already stated.

Kühne has not represented the capillaries in any of his drawings. The explanation of the appearances represented by him in the nervenhügel, is seen by reference to my fig. 8, Plate XXIII., Phil. Trans. 1860, in the upper part of which is just such a collection of nuclei with the nerve-fibres, in which they

are imbedded. In Kühne's drawings the fine nerve-fibres are disintegrated, and the nuclei completely disarranged.

The appearances represented in these 'nervenhügel' of Kühne were familiar to me in the mouse, before I arrived at the conclusions published in my first paper, and at this time I succeeded in demonstrating that the arrangement of the 'hügel' was not natural.

In Plate XI., fig. 9, the points to be seen upon the surface of the sarcolemma of a muscular fibre from the diaphragm of the white mouse are represented, and in fig. 10 a portion is shown much more highly magnified. The nuclei are connected with nerve-fibres, which lie outside the sarcolemma. In fact vessels and nerves can be stripped entirely away from the sarcolemma of the muscular fibre of the white mouse, leaving this membrane entire. Had this specimen been prepared according to the methods in ordinary use, all that would have been observed would have been a cluster of nuclei, irregularly arranged, like those in the nervenhügel.

Further, the mode of formation of the nervenhügel, so distinct in the insect muscle, will be seen by reference to my paper, just published in the Microscopical Journal.*

But it must not be supposed from these remarks, that I altogether deny the existence of the expansion of the nerve over the elementary muscular fibre, as delineated in this and in Kühne's last paper. Far from this, I readily acknowledge that such an appearance does exist, and I have beautiful specimens showing distinctly some of the appearances in question, but the structure is undoubtedly outside the sarcolemma, and my interpretation is very different from that given by other observers who have seen the appearance in question,—but this shall be fully considered in another memoir.

In the paper, "Die Muskelspindeln" (Virchow's Archiv., vol. xxviii.) Dr. Kühne has ventured to represent a dark-bordered fibre, close to its supposed intramuscular termination, three-fourths as wide as the muscular fibre itself! Part of this figure I have copied in Plate XI., fig. 8. In a former paper he has represented as one nerve a bundle of fibres, which really crosses over or under the muscular fibre, and probably divides into smaller bundles of finer fibres, which pursue a course of some length before they reach their ultimate distribution.

Kühne, however, supports his former opinion in yet another

* On the structure and formation of the sarcolemma of striped muscle, and of the exact relation of the nerves, vessels, and air-tubes (in the case of insects), to the contractile tissue of muscle. Trans. of the Mic. Society. See also Figures 4 and 5, Plate XV., of this journal.

paper (Der Zusammenhang von Nerv und Muskelfaser, Virchow's Archiv., vol. xxix). The illustrations accompanying this paper represent the nervenhügel and their connection with the muscular tissue, as seen in a transverse section of the elementary muscular fibres of the white rat and mouse. I have had these figures copied in Plate XV., figs. 1 and 2. They explain themselves. I may remark, with reference to these figures, that in my own specimens from the white mouse, the dark-bordered fibres certainly do not terminate as Kühne has represented, and that exceedingly pale granular fibres can be followed for a considerable distance beyond the point where the highly refractive white substance ceases (see figs 9 and 10, Plate XI., in which the capillaries are also represented).

In Plate XV., fig. 3, I have copied (but reduced to half the size) Kühne's last representation of the arrangement of the nerve plates and the formation of the nervenhügel. The axis cylinder is here made to expand into a broad 'end plate.' The oval nuclei near the expansion of the axis cylinder are the nuclei of the 'nervenhügel.' The nuclei of the sarcolemma are marked. I must beg the reader to compare the nerve 'end-plate' in this figure with the terminal pale fibres, &c., 'nerven-end-knospen' figured in Plate XI, fig. 1, published in the last number of the Archives. (Taf. ix. Virchow's Archiv., Vol. XXX.)

The reader will have noticed, that there is a broad and essential difference between continental observers and myself in reference to the distance which fibres forming the structural continuation of the dark-bordered fibre may be followed beyond the point where the dark-bordered fibre ceases. Kühne, Cohnheim, Rouget, Krause, Engelmann, make the nerve end a very short distance from the point where the dark-bordered fibre ceases, and the latest researches make the dark-bordered fibre end abruptly in its terminal end-plate, so that there is not even a pale fibre as long as that of the Pacinian corpuscle. Kölliker, on the other hand, traces pale fibres for some distance in continuation with the dark-bordered fibre and on the external surface of the sarcolemma. So far, in essential points, Kölliker coincides with me, but I stated that the fibres did not end as Kölliker supposed, but might be followed for long distances as excessively fine fibres only demonstrable, however, in specimens prepared in a particular manner.

It was to be supposed that these pale fibres which I have described as nervous, would be characterised as fibres of connective tissue, by opponents who have not seen my specimens, and especially by those who have not examined my drawings, or

carefully perused my observations. This is at the same time the obvious and the most easy mode of attack, since it is not possible for me to show these fibres to every one, and any assertion I may make with regard to them, such, for example, as, 'that I have traced them onwards from the dark-bordered fibre,' may be doubted and set down as an error of observation or as resulting from the mode of examination employed, &c. For years it was maintained that 'Remak's fibres' were connective tissue, and the doctrine is still held by some German observers. There are persons who look upon branches of the sympathetic as modified connective tissue, in which 'apolar' nerve cells are embedded, and who do not consider what such fibres are for, or what purpose is fulfilled by such cells. There are observers who seem to think that connective tissue is the most important texture in the body, that it has special canals for its nutrition, special corpuscles for its formation, that it supports higher tissues in position, that it is the principal, if not the sole, seat of some of the most important pathological changes.

It has often happened that the mere dictum of one observer has been permitted for years to outweigh the facts ascertained by the actual observation of others, and it is natural, and, indeed, right, even in matters of mere observation, that a high authority having a well-earned position as an observer and a high reputation as an author, should influence the opinions of others, and, indeed, exert very considerable weight.

At the same time it will be conceded by the highest authorities themselves, that good observers have been led to entertain erroneous views, and there is no department of natural knowledge in which such conflicting statements have been made upon mere matters of fact and observation as in minute anatomy. I must therefore press my own observations upon the attention of observers, not as having authority or reputation, but simply as an observer who has at least spared no time, pains, or trouble during many years to ascertain the truth regarding the mode of distribution of nerves to muscle.

In several papers and in many drawings I have endeavoured to show that the fine fibres I have described as nerves are structurally continuous with dark-bordered fibres. I have stated that the specimens may be examined by any observers who desire to see them. I have shown that fine fibres agreeing in every respect with those I have described in muscle, exist in other tissues to which nerves are distributed, and many of these are admitted by all who have seen them to be nervous. In Plate XIII., fig. 1, at *d*, is a fibre which would be called con-

nective tissue by some, continuous with a dark-bordered fibre. In fig. 2 are 'connective tissue fibres' (?) running on one side only of a dark-bordered fibre, giving off branches which further divide and sub-divide and form a plexus or network of very fine "connective tissue fibres!" In the other figures in this plate fibres of the same kind are seen, and their continuity with undoubted nerve fibres, either at the spot copied in the drawings or at a short distance from this, was demonstrated. Corresponding fine fibres are represented in Plate XIV.

Then there is collateral evidence in favour of the conclusions, which would, I think, be drawn by any unprejudiced person from a careful examination of several of my specimens, and yet, in spite of all, Kühne reiterates, the old cry, 'connective tissue,' 'vascular nerves!' No arguments are advanced, no specimens to explain the facts in another way made, nay, specimens are not even prepared, according to the method I have stated to be necessary, to see the fine nerve-fibres with the very extensive distribution I have described. While it is certain that plexuses and networks exist, is it not possible that 'Endplatten,' 'Nerven-end-knospen,' 'Nervenhügel,' &c., may exist as well (?), but Kühne is not contented with establishing the existence of the structure he has described, but proceeds to state that networks and plexuses described by me are not to be demonstrated, although I have seen them, and have shown them to other observers.

I would ask:—will observers accept the conclusion, that long elementary muscular fibres are influenced by the nerves at only one single point in their length, when dark-bordered and fine fibres cross them at short intervals throughout the entire length of the muscular fibre, as any one can demonstrate in the muscles of the eye of the frog, or, better, the cameleon, or in the diaphragm of the mouse, or in the very thin mylohyoid of the frog? For what purpose do the nerves cross the muscular fibre at many different points and pursue a most circuitous course, if a nerve-fibre is to become connected with it at one spot only, and influence the muscular fibre at this single point?

Those who hold that the plexuses of coarse and fine fibres which are to be demonstrated in the case of every muscle are an arrangement not connected in any way with the action of the nerve upon the muscle, should show not only why nerves invariably exhibit this arrangement, but why the nerve-fibres do not at once pass to the point of the muscular fibre with which they are connected, by the shortest and most direct route.

Will observers accept the conclusion that the relation of

nerves to striped and unstriped muscular fibre is essentially different, and that the unstriped fibre receives a more abundant supply of nerves than striped muscle? Will anatomists accept the fact that nerves in tissues generally form networks and plexuses of exceedingly fine fibres, without doubt,—and the statement that in this one tissue—voluntary muscle, they terminate in end plates in contact with the contractile tissue? Is it probable that the muscular fibres of the heart should be supplied with nerves upon a principle totally different to that which obtains in other striped muscles, or that some muscles of the tongue should be supplied by nerve networks, and others by nerve plates as must be the case, unless it be shown that the finest muscular fibres in the tongue, the diameter of which is many times less than that of the smallest nerve-plate figured, nay, less than the terminal portion of many of Kühne's dark-bordered fibres—are not supplied with nerves at all, although they contract like other striped muscles?

Will it be accepted as a fact, that the terminal nerve-fibres in the muscles of the trunk and limbs are many times the diameter of the terminal fibres in other tissues, and that a very long muscular fibre is influenced in its entire length by an end organ at one or other extremity, or in its centre? Can it be supposed that it is necessary for the nerve tissue to come into absolute contact with the contractile tissue of the muscle while nerves are admitted to influence other tissues without being in structural continuity with them?

Will observers accept the conclusion that the nerve perforates the sarcolemma of voluntary muscle before any observations have been offered upon the manner in which it is developed in such a locality? If the views which are now advocated by Kühne and others, with regard to the arrangement of nerves in muscle be true, it must follow that muscle forms a special exception to every other tissue in the body, both as regards the mode of distribution of its nerves, and the manner in which nerves influence the tissue. Will anatomists accept these views as affording the true account of the distribution of nerve to voluntary muscle, in the face of the fact that neither the vessels nor the nuclei connected with them are to be seen in the specimens advanced as demonstrative of the doctrines most in favour in Germany? Is it probable that a plan of demonstration which fails to render the vessels and other structures distinct and clear, should render definite a structure of such far greater delicacy than vessels, as the pale nerve-fibres? On the other hand, is it not reasonable to conclude that a plan of demonstration, which it is admitted displays most clearly the

finest nerve-fibres in tissues generally, will enable us to demonstrate them in muscle?

The arrangement of the terminal branches of the nerve in voluntary muscle advanced by Kühne and others, if true, is peculiar to this tissue alone. There is no other tissue in which nerve-fibres have been shown to terminate in end plates of such remarkable structure, and the most positive demonstrative evidence is required before such a deposition can be accepted as the normal and essential arrangement necessary for the action of the nerve upon the muscle, in the face of collateral evidence of the strongest character.* This collateral evidence, and the positive observation in innumerable specimens of a very different arrangement, are met by the mere assertion that the nerve-fibres seen are not nerve fibres, and are composed of gelatin, or if nerves, they are vascular nerves.

I would answer simply that the fibres in question are of the same physical and chemical character as the fibres admitted to be n e r v o u s in the cornea, the bladder and mesentery of the frog, and elsewhere. They are structurally and directly continuous with dark-bordered fibres. Moreover, in the same specimen, the vessels, the vascular nerves, and the motor nerves are all demonstrated, and not only so, but the course of the fibres can be followed over a very wide area, and, oftentimes, fibres as fine as those in the cornea can be followed from one undoubted nerve-fibre, across many elementary muscular fibres to another trunk.

With reference to the points urged by Kühne and those who support him, I would remark that no answer has been given to the obvious objections that may be made to the interpretation he has given of the appearances he has seen. For instance, it may fairly be said—

1. That the nerves he gives as penetrating the sarcolemma really lie over or under the muscular fibre he delineates.†

2. That the irregular arrangement of the nuclei represented by him results from derangement, and does not exist in nature.

* I wish it to be clearly understood that I do not deny that the appearances described have been seen and are to be demonstrated, but I think that they have been probably interpreted wrongly, and I am led to the conclusion, which is supported by very strong evidence, that the view in question does not represent the mechanism through which nerve as a general rule influences striped muscle.

† I have remarked that one very important advantage gained by the use of very high powers ($\frac{1}{25}$ and $\frac{1}{50}$ magnifying from 1,500 to 3,000 diameters linear) is that structures apparently upon the same plane are demonstrated to be on very different planes. In muscle a nerve-fibre often appears by a twelfth to be situated beneath the sarcolemma, while the $\frac{1}{25}$ or $\frac{1}{50}$ show it to be situated beneath the muscular fibre itself.

While it is to be remarked—

3. That he has never seen the fine branches of the nerve-fibres which are to be demonstrated by other processes of investigation.

4. That many of the nuclei considered by him and others as nuclei of the nerve-sheath are really the nuclei of fine nerve-fibres which run in the sheath. These fine nerve-fibres are figured in several of my figures. I would especially draw attention to figs. 1 and 2, Plate XIII.

I will here record the brief criticism passed upon my observations by a French anatomist, M. Rouget, "Il est facile de s'assurer que la description de Beale est completement inexacte et ne saurait arrêter longtemps un observateur exercé et attentif." (Brown-Sèquard's Journal, 1862)!

It would be superfluous to reply to these remarks, which not only settle the question at issue, but render nugatory any conclusions to which I may be led by future work. All that remains for me is to commend the observations of M. Rouget to the attention of every 'observateur exercé et attentif.'

It is unnecessary for me to reply specially to the views of Krause, Margo, Colnheim, Aeby, Engelmann and others, for the remarks I have offered really form an answer to their conclusions, which, like Kühne's, are completely at variance with mine. Moreover, Kühne has recently given an outline of their conclusions in the 30th volume of Virchow's Archiv. The sketches illustrating his remarks are rather rough, and the one he has inserted as an illustration of my view is likely to cause rather an erroneous impression as to the course actually taken by the nerve-fibres as they traverse the muscles. This diagram is rendered still more confused by the attempt to give Kölliker's view as well as my own in one drawing. In a controversy of this kind observers should endeavour to give accurate representations of the views of their opponents. If my friend Kühne refers to my conclusions at all, it is but just he should give his readers a clear account of the views which I hold.

I now propose to offer some further remarks upon matters of importance already referred to in my memoirs, but which have not been noticed by those who have criticised my observations. Some of these observations seem to me of considerable importance, because they afford valuable collateral evidence in favour of my general conclusions.

Of the fine Nerve-fibres of the Sheath.

The nuclei represented by Kühne and Kölliker as the nuclei

of the sheath of the nerve, are clearly seen in my specimens, mounted in highly refracting fluids, to be nuclei of fine nerve-fibres running in the sheath. Kölliker, in some of his drawings, has represented the dark-bordered fibre as ending within the sheath, while the latter is prolonged onwards for some distance, and then made to terminate abruptly, or in a more or less rounded extremity.

The different and incompatible views existing between continental observers and myself are in some measure due to this sheath question. The so-called sheath is not a 'tube' or 'membrane,' or 'tubular membrane,' which contains the other constituents of the nerve-fibre; nor is it a sheath which invests them, but it is simply a transparent matrix, in which nerve-fibres, coarse and fine, are embedded. The so-called sheath is not formed as a special structure to invest the nerve-fibres, but it results from changes occurring in the nerve-fibres themselves. This 'sheath,' or 'tubular membrane' of the so-called dark-bordered fibre precisely corresponds to the transparent connective tissue, in which the fine nerve-fibres are imbedded. It is a form of connective tissue, and in many situations where nerves existed at an earlier period, nothing but this so-called sheath remains. All the soluble fatty matters have disappeared, and this material, which is not readily absorbed, is left behind. Vessels may waste, and ducts of glands may waste, and leave behind the same sort of transparent connective tissue. Moreover, as I have before stated, it is altogether a fallacy to suppose that near the peripheral distribution, every single branch of nerve-fibre is surrounded by its own separate sheath. The fact is, that several branches are imbedded in the same band or cord of transparent connective tissue, and fibres from one side of such a band often cross over to the opposite side, and in very many instances one fibre pursues a spiral course around another.* There may be one or two dark-bordered fibres, and five or six very fine fibres, embedded in the same matrix (Plate XIII., fig. 3). The fine fibres, in many cases, may be most positively proved to be continuous, with dark-bordered fibres. It is almost certain that the great majority of these 'fine fibres of the sheath' are continuous with dark-bordered fibres. A very distinct instance of this is represented in Plate XIII., fig. 1.†

* See "New observations upon the structure and formation of certain nervous centres," 1864. Plate VI., Fig. 29.

† The engraver has, unfortunately, cut away from fig. 3, Plate XI., several of the fine lines, regarded by me as fine nerve fibres. The power under which this specimen was examined (700 diameters) is too low to see them very distinctly in many specimens, and it often happens that these delicate fibres are destroyed in preparing the specimen.

Doubtless some of my opponents will still cleave to the idea that these fine fibres in the sheath are not nerve-fibres at all, but the evidence is most conclusive, and I am quite ready to discuss the question at a greater length than I have done hitherto. I will only say here, that if the fine fibres which I have figured are not nerves, it follows absolutely that there are no fine terminal nerve-fibres at all, and all nerve-fibres terminate in connective tissue. The view that no fibre is a nerve-fibre unless it be dark-bordered, cannot any longer be entertained, for in no peripheral tissue or organ that I have studied does the really active part of the nerve-fibre commence, until the dark-bordered character has ceased. Where the dark border ends is not where the nerve ends, but it is where the extensive active peripheral nerve plexus commences. If all the fine plexuses demonstrated in muscle are not nervous, the plexuses of precisely the same character in the bladder of the frog, in the cornea, and other tissues, admitted by my opponents to be of this nature, are not nervous. I conclude no one would maintain that neither the fine fibres I have described in muscle, nor those in the bladder, &c., are nerve-fibres.

It has, then, been shown that at least in very many cases the fine granular fibre which invariably accompanies the fine terminal portion of the dark-bordered fibre is continuous with a dark-bordered fibre in the trunk.

This is the case in motor nerves distributed to muscle (pectoral, mylohyoid; hyla), and also in the nerves distributed to sensitive surfaces (palate, frog).

And it has been shown that a fine dark-bordered fibre often divides at a certain point into *a*, a dark-bordered fibre, and *b*, a bundle of fine fibres.

It has been shown that dark-bordered fibres seldom run alone. I have so seldom seen a fine single dark-bordered fibre which has not been accompanied by a pale, granular fibre, which invariably pursues a spiral course round it, that I am inclined to think that all dark-bordered fibres near their destination are accompanied by fine fibres.

And it has been shown that at the peripheral distribution fine granular fibres (probably composed of several finer ones) do run alone, that is without dark-bordered fibres, forming networks. Many of these very fine, pale, granular fibres are not more than the $\frac{1}{100000}$ of an inch in diameter, but there is reason to conclude that even these are composed of still finer fibres.

And it has been shown that the fine granular fibre running by the side of the dark-bordered fibre in certain cases unites

with the bundle of fine fibres, forming the continuation of the latter, to form a common, fine granular trunk, composed of very many fine fibres.

From many facts demonstrated, it seems probable that nerve-fibres, as they pass to their distribution, divide into a vast number of separate branches, which, after pursuing a course over the tissue to which they are distributed, of some extent, pass into other nerve trunks, and thus return to the cells of the nerve centre from which they started.

Nerves Distributed to the Elementary Fibre throughout its Length.

It should be particularly borne in mind that the differences between Kölliker, Kühne, Margo, Engelmann, Rouget and many others, and myself, with reference to the supply of nerves to voluntary muscle are very wide. Although the above observers differ from each other as to the precise relation the nerve bears to the contractile tissue of the elementary fibre; they all agree that the nervous supply to each individual elementary fibre is limited to a very small portion of its entire extent. Some indeed think it doubtful even if every fibre of a muscle is supplied at all, believing that only a small part of the entire muscle is brought into any very near relation with the nerve-fibres. Now, although I have not demonstrated the fact so distinctly as it is doubtless capable of being demonstrated, upon the large elementary muscular fibres of the frog, I am convinced that even in them there exists a network of very fine nerve-fibres, with nuclei here and there all over the surface of the sarcolemma, from one end of every elementary muscular fibre to the other. Doubtless, in some portions the meshes are finer than in others, and the fibres are much more numerous, but everywhere very fine but faint, and exceedingly delicate fibres are occasionally to be demonstrated. They much resemble the fine fibres in the bladder, in the cornea, and the mesentery of the same animal.

Nor have the above observers described the very numerous dark-bordered nerve-fibres which cross the muscular fibres at comparatively short intervals, as, for example, in the muscular substance of the tongue, the little muscles of the eye, over which there may be seen a complete network of dark-bordered fibres (remarkable in the muscles of the eye of the cameleon).

I have repeated my observations upon the muscles of the white mouse, and feel certain that the representations I have given are correct, although the execution of the drawings is

somewhat too rough and coarse to give a correct idea of the actual appearances observed.

It should be remembered that in these specimens the vessels are injected, and the capillaries form a very fine network over the surface of the muscular fibres. As I have stated, in the intervals, very fine granular-flattened nerve-fibres may be seen. Nuclei can be seen in the capillary walls, and nuclei in the fine nerve-fibres, and, besides these, the nuclei of the muscle. In fact the elementary fibres of the diaphragm of the white mouse are almost entirely covered with nerves and capillary vessels.

Opponents have asserted that the nuclei I have described are the nuclei of the connective tissue. In reply, I affirm that not only is much of their 'connective tissue' composed of nerve fibres but they have mistaken the nuclei of capillaries, as well as those of nerves, for connective tissue corpuscles.

Of the Sentient Nerve-Fibres of Voluntary Muscle.

It has been inferred by Reichert, and the opinion has been supported by Kölliker, that certain nerve-fibres distributed for the most part upon the surface of the flat thin breast-muscle of the frog are sentient in their nature. These so-called sentient fibres are stated by the above observers to be derived from one principal 'sentient fibre,' in the same trunk with the purely motor nerve-fibres, and Kölliker states that these sentient fibres branch out over the whole extent of the muscle, and may be seen even in parts which are destitute of 'motorial nerves.'

What I have been able to observe, with regard to the distribution of the bundle of dark-bordered fibres upon the breast-muscle of the frog, does not convince me that the above view regarding the sensitive function of these fibres is true. What I have seen is as follows :—

The bundle of nerve-fibres passing to the breast-muscle, as it crosses the muscle divides into several smaller bundles resulting from the subdivison of the primary nerve-fibres which pass to different parts of the muscle, from these minor bundles many nerve-trunks pass, which at length divide and subdivide upon the surface of the sarcolemma of the nearest elementary muscular fibres, while some pursue a much longer course, passing between muscular fibres, or ramifying in the connective tissue upon the surface of the muscle. There are in fact—

1. Dark-bordered fibres which divide upon the surface of the sarcolemma, and in contact with it, and,

2. Dark-bordered fibres which may be traced over and amongst very many muscular fibres for a long distance.

The fibres of the first set appear to lose themselves upon the sarcolemma, the last often seem to be lost amongst the connective tissue. The first are regarded as purely motor, the last are the supposed sensitive nerves of the muscle. Both kinds of nerves result from the subdivision of dark-bordered fibres which enter into the formation of the large bundle which is distributed to the muscle. But the so-called sensitive fibres may be traced a very long way from the large nerve trunk, and many of their finest subdivisions, of $\frac{1}{5000}$ of an inch in diameter, or less, may be seen to run on in company with other nerve branches, and can be followed into trunks composed of fine fibres, which pass away from the muscle at different points of its borders, some passing to the cutis, some to the connective tissue with which the muscle is continuous at the inner and outer edge, and others issuing from the muscle near its insertion into the fibrous tissue which gives origin to the abdominal muscles.

But some of the so-called sensitive-fibres are seen to pursue a still longer course. Even from these, branches are given off which may be followed to muscular fibres, and may be seen to divide upon the surface of the sarcolemma in precisely the same manner as is observed with regard to dark-bordered fibres which result from the immediate division of the large fibres constituting the original bundle (Plate XI., fig. 3).

The general course and distribution of the nerves to the pectoral of the frog are the same in the young, fully-developed, and old frog, except that as age advances, the fibres which are followed to, and seen to be distributed in, the connective tissue covering the muscle increase in number. In very young frogs, as far as I am able to observe, all the fibres resulting from the division of the primary fibres ramify upon elementary muscular fibres, or are to be traced to the vessels of the muscle. At this period the thick layer of connective tissue present in the adult is so thin that only indications of it can be discovered.

All these so-called sensitive fibres cannot be traced into, or shown to result from, the division of one or two of the dark-bordered fibres of which the bundle is constituted, as stated by Kölliker. So that there is nothing in the arrangement, course, or destination of these fibres to lead us to consider them sensitive fibres, and until new facts are brought forward in

favour of the opinion entertained by Reichert and Kölliker, it can scarcely be accepted.

1. It is certain that some of the branches of the supposed sensitive fibres of the trunk are lost on the sarcolemma and are distributed in precisely the same way as the fibres regarded as purely motor.

2. That the branches from which nerves distributed to the sarcolemma are derived, also give off fibres which are lost in the intermuscular connective tissue or in that upon the surface of the muscle—that is, the fibres considered to be purely motor give off some of the branches regarded as purely sensitive. And hence it results that all the branches considered by Reichert and Kölliker to be purely motor are not so, nor are all the fibres regarded by them as sensitive of this nature.

It remains to be proved if any of the fibres ramifying in the connective tissue upon and amongst the muscular fibres are sensitive. My own opinion is that there are no special sensitive fibres distributed to the muscle, and that the fibres in the fully-formed muscle considered to be of this nature are motor fibres, some of them probably having supplied at an earlier period of life muscular fibres, which have since disappeared, and these nerve-fibres now seem to be distributed to the connective tissue covering the muscle. I think it probable that the only fibres which have the power of transmitting sensations from the muscle to the nervous centres are those which are distributed to the capillary vessels, or some of those filaments which leave the muscle and pass into the bundles of cutaneous nerve-fibres.

Of the "Nerve-tufts" of the Breast Muscle of the Frog.

The question concerning the arrangement of the nerve-fibres in voluntary muscle receives elucidation from a study of the nerves distributed to the so-called 'nerve-tufts' of the breast muscle of the frog and figured by Kölliker (Proceedings of the Royal Society, May 1st, 1862, p. 78). This figure I have had copied (see Plate XVI, fig. 1). It does not, however, represent all that is to be seen in these swellings, for the numerous oval nuclei, figured in Kölliker's drawing, are represented by him as being pretty generally diffused throughout the swelling, and as not connected with one another or with any definite structure, nor is the relation of the nerve-fibres to these nuclei indicated by Kölliker.

Some of these nuclei are, however, seen in my specimens to be nuclei in the course of very fine nerve-fibres—nuclei which

take part in the formation of the nerve-fibres themselves. Others are the nuclei of the muscular fibres which are undergoing development, over the surface of which the fine nerve-fibres are spread out. These facts are demonstrated in several specimens which I have mounted in strong glycerine and acetic acid syrup.* A portion of one of these is represented in Plate XVI, fig. 2. This drawing which is magnified 700 diameters appears somewhat confused owing to the very close proximity of the nerves and muscles. It is, however, a careful copy of one of my specimens just at the spot where three dark-bordered nerve-fibres pass into or emerge from one of the 'nerve tufts.' The 'explanation' of this figure should be referred to. In fig. 3 I have shown one branch of a dark-bordered nerve-fibre and its division into the very fine fibres. These may be followed for a considerable distance amongst the developing muscular fibres.

It has been truly stated by Kölliker that the apparently single muscular fibre bearing the swelling is really a bundle of very fine fibres, varying from three to seven, or more, in number, and that the apparently p e n e t r a t i n g nerve-fibres merely pass between these imperfectly developed muscular fibres. I cannot however agree with him in the view that the fine muscular fibres result from longitudinal splitting of the fibres. The bundles of fine muscular fibres under consideration, extend at a certain period of their development from one extremity of the muscle to the other, but all the muscular fibres of the bundle do not reach so far. In one bundle sometimes ten or twelve distinct muscular fibres very closely packed together may be counted. Near the swelling the muscular fibres are wide, and many often terminate in fine pointed extremities. In fact at this point several spindle-shaped, nucleated, transversely striated muscular fibres may often be seen, and the stages through which the elementary fibres of voluntary muscle pass in their development may be traced.

In these "nerve tufts" we may indeed study in the fully-formed animal, striped muscle and nerve in every stage of development. Vessels cannot be traced into the youngest tufts, but in those which consist of several partly grown muscular fibres capillaries are to be seen, so that the development of muscles, nerves, and vessels can be studied in these imperfectly developed "tufts."

From the above observation, it will be seen that I cannot

* I have found this strong acetic acid syrup a most valuable agent in these and kindred investigations.—"How to Work with the Microscope," third edition, p. 202.

agree with Kölliker in the view he has taken of these bodies. He says, "Now if it be admitted that the finer muscular fibres composing the bundle are generated by the division of thicker muscular fibres, as Weismann justly concludes, the explanation of the nerve tufts becomes easy, inasmuch as they may be conceived to arise from a simultaneous growth and division of the nerve fibre belonging to the parent muscular fibre, in order that each of the young muscular fibres may obtain its branch of nerve" (Croonian Lecture, May, 1862).

Kölliker thinks that the narrow young muscular fibres result from the division of old ones. So far from this being the case, the young muscles and young nerves are developed from collections of nuclei or masses of germinal matter, precisely resembling those which are found in the embryo. If new muscular fibres are to be developed from old ones, the 'nuclei' increase in number, the old muscular tissue undergoes disintegration, and is absorbed, and a mass consisting entirely of nuclei, or masses of germinal matter, by the agency of which the formed material of the new fibres is at length produced, results.

As I have pointed out on many occasions, in fully formed organs there exists a certain proportion of embryonic germinal matter, which undergoes development at a future period of life, and if this becomes fully formed tissue, still there remains embryonic matter for development at a still later period, and so on. In the situations of these so-called nerve tufts in the breast-muscle of the frog, new elementary muscular fibres are added to those already formed, and the muscle grows as the frog advances in age. And in the formation and growth of the muscular fibres, and in the formation and arrangement of the nerves around them, the movements of the several nuclei or masses of germinal matter to which I have drawn attention, play no unimportant part. (See my paper on 'The movements of the living or germinal matter of the tissues of man and the higher animals.' Archives, Vol. IV., page 150).

With reference to the nerves supplying these so-called nerve tufts, I would remark:—

1. That two dark-bordered nerve-fibres, running in the same sheath, may often be traced to one part of the 'nerve-tuft.'

2. Besides the dark-bordered fibre or fibres, there are invariably very fine fibres running in the same sheath.

3. That the dark-bordered fibres and the accompanying fine fibres divide and subdivide very freely amongst the young muscular fibres, and that thus quite a leash of very fine nerve-fibres results, in the course of which numerous nuclei exist at certain intervals. Many of these can be followed upon or between the

muscular fibres, for the distance of the twentieth of an inch or more from the oval swelling (see fig. 5, Plate XIII).

4. That the dark-bordered fibre or fibres which enter at the tuft are not the only nerve-fibres distributed to these bundles of muscular fibres, but that invariably a bundle, consisting of two or three fine, but dark-bordered fibres, is connected with the muscular fibres, at a point above or below that at which a swelling is situated, where the large fibre or fibres enter. Sometimes there are two such bundles, one above and one below. These not unfrequently give off branches, just before they pass to the muscular bundle, which pursue a longer course, and are distributed to other larger muscular fibres, and oftentimes branches pass from one muscular bundle to more distant ones.

From the above observations it follows, that these 'nerve tufts' in the breast muscle of the frog, consist of developing muscular fibres, which are freely supplied with nerves, and that the number and distribution of the nerves render it probable, not only that there are entering and emerging fibres, nerve loops, and plexuses, or networks upon the muscular fibres, rather than free ends, but that the contraction of the new muscular fibres are harmonised with those of the other and older elementary muscular fibres of the muscle by commissural branches of nerve fibres.

I think it not improbable that in some cases the organs termed 'end plates' are of the nature of these so-called nerve tufts in the breast muscle of the frog, that is, that so far from being special organs in connection with one individual muscular fibre upon which they are found, they consist of masses of imperfectly developed structure—of tissues in an embryonic state, which would have undergone development into fully formed nerves and muscles at a future period of life. Certain it is, that all these 'end plates' are external to the sarcolemma; that into every one that I have seen more than one nerve-fibre, and oftentimes as many as six or eight may be traced, and that they are never placed, as has been represented, at the end of a long branch of nerve-fibre, but are always appended as it were, to a nerve trunk, crossing numerous muscular fibres, and invariably composed of more than two fine nerve-fibres. Oftentimes nerve-fibres may be seen passing away from the 'end organ.' I do not, however, mean to imply that these apparent end organs are all of this nature. Some undoubtedly very closely resemble in their arrangement some of the nerves upon the elementary fibres of the breast muscle of the frog, and like these lie upon the sarcolemma, and are adherent to it. Into every one that I have seen, more than one nerve-fibre passes. These 'end organs'

are very distinct in the muscles of many lizards, and especially in the cameleon; I propose to publish drawings of the 'end organs' of the latter animal shortly. Kühne's latest figure of these end organs has been copied from his paper published in the 30th volume of Virchow's Archiv., 1864, and is represented in Plate XV., fig. 3, reduced to half its size. Kühne asserts that the structure represented lies beneath the sarcolemma, and one terminal fibre passes into it. As I have recently prepared some very good specimens of these bodies, I propose to discuss the question fully in a separate memoir.

Further Observations on the Nerven-hügel.

In the Comptes Rendus for November 21, 1864, M. Rouget endeavours to show that Kühne's Doyèreschen Nerven-hügel in the muscles of certain invertebrata result from the sarcolemma being drawn up in a cone, as the nerve which adheres to it is stretched, and states that, at the nerven-hügel, the nerve-fibre divides into two fine fibres, which may be traced for some distance, and then terminate. "Leur extrémité terminale est légèrement effilée; elle ne présente ni plaques, ni noyaux, ni substance finement granuleuse."

The structure of these nerven-hügel in insect muscles was fully described by me in a paper, accompanied by several drawings, read to the Microscopical Society on June 8th, 1864, and published in the 'Transactions' on October 1st, 1864, or three weeks before the appearance of M. Rouget's memoir. Rouget confirms my statements with reference to the production of the nerven-hügel, but we are at variance upon the further progress and mode of termination of the nerve-fibre, Rouget maintaining that it penetrates beneath the sarcolemma and terminates in a very fine fibre, in contact with a very limited portion of the contractile tissue, while I have been able to trace the nerve for a long distance beyond the point at which he makes it end, dividing into very fine fibres, which form an extended network upon the sarcolemma. Rouget's researches, however, as far as they have gone lead him to conclude that the arrangement of the nerves in the muscles of articulata is totally distinct from that met with in vertebrata. "Il résult de ces faits qu'il n'y a pas identité entre les divers modes de terminaison des fibres nerveuses motrices chez les vertébrés et les articulés." On the other hand my observations lead me to the conclusion that the arrangement is in its essential points the same in all. In no case are there nerve-ends, but always plexuses, or networks,

which are not in structural continuity with the contractile tissue of muscle. In no case is the distribution of the nerve-fibre confined to a very limited extent of muscle as is maintained by most continental anatomists.

I trust that in future all writers upon this question will give drawings of the appearances they have observed, for mere description without drawings can do nothing towards clearing up the many conflicting statements.

The Demonstration of the actual Arrangement of the very fine Nerve-fibres upon the Sarcolemma of the Elementary Muscular Fibres.

I have already described the arrangement of the finest nerve-fibres in the mylohyoid of the hyla, and have pointed out that in this muscle fine fibres may be actually traced from one undoubted nerve-trunk to another (Proceedings of the Royal Society, June 5th, 1863). The plate illustrating this paper is reprinted in Plate XII.

I have recently succeeded in preparing a specimen of the breast-muscle of the frog, in which the arrangement I had inferred must exist from the facts recorded in my earlier papers, is actually demonstrated, and the fine fibres resulting from the division of the dark-bordered fibres, and the fine fibres running in the sheath become continuous with a network of very fine fibres ramifying upon the surface of the sarcolemma, and, as it were, beneath the connective tissue. At intervals nuclei are seen as in other cases. In fact this plexus or network upon the surface of the sarcolemma of the muscles of the frog very closely resembles, in its general arrangement, the network of nerve-fibres in the heart, tongue, bladder, cornea, and connective tissue of the same animal. Its extreme delicacy renders it very liable to destruction, and as the thickness of the elementary muscular fibres, and the numerous lines existing in their structure interfere with the passage of the light, it is not surprising that there should be great difficulty in demonstrating it.

The nerve-fibres constituting this plexus, although compound, are so fine that they are quite invisible unless very high powers be employed, and the specimens specially prepared. In recent specimens of muscle not the slightest indication of the extensive system of fine fibres which I have described is to be seen, nor is the muscle peculiar in this respect, for the finest nerve-fibres cannot be seen in recent specimens of the frog's bladder, pericardium, connective tissue, &c. So pale and

delicate are these fine nerve-fibres that they cannot be seen even in the most delicate connective tissue until the latter has been rendered transparent, and the nerve-fibres themselves granular. The nuclei in connection with them are lost amongst the numerous nuclei of the connective tissue, in which the nerve-fibres lie. By the slow action of acetic acid upon specimens immersed in glycerine or syrup, the fine nerve-fibres are decomposed and rendered granular, in consequence of minute oil globules being set free. This change has enabled me to trace the course and distribution of the finest nerve-fibres in a great many different tissues.

By proceeding as I have elsewhere fully described, the appearances to which I have drawn attention may be demonstrated.*

I claim, therefore, to have demonstrated that nerve-fibres passing to a muscle divide at length into a vast number of exceedingly fine pale granular fibres, which ramify upon the external surface of the sarcolemma, connected with which fibres at certain intervals, are oval nuclei (Phil. Trans., 1860), and that these fine fibres after an extensive and in many cases very circuitous course, join with other fibres to form dark-bordered fibres, which at length pass towards the nervous centre either in the same bundle as the dark-bordered fibres passing towards the muscle or in other bundles.

It therefore follows, that of the dark-bordered nerve-fibres distributed to a muscle, some pass from the nervous centre towards the muscle, some from the muscle towards the nervous centre, and that the nerve-fibres do not end in the muscle at all. The results of these researches, considered in connection with those arrived at from the investigation of various kinds of nerve centres, justify the inference that the fundamental arrangement of a nervous apparatus is a complete and continuous circuit.

My position in this enquiry is as follows:—

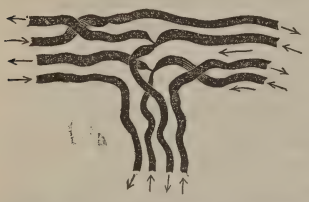
1. I was led to the conclusion of the existence of a network of nucleated nerve-fibres, many, if not all, composed of finer fibres upon the surface of the sarcolemma of the muscles of the mouse in 1859, the observations being published in the Phil. Trans. for 1860.

2. That after having studied the conclusions arrived at by Kühne and Kölliker upon the breast muscle of the frog, and those of other observers opposed to my own views, I have, after working for a very long time upon the same muscles employed

* How to work with the Microscope. 3rd Edition, 1865.

by them, succeeded in tracing the nerve-fibres a much greater distance from the dark-bordered fibres, and have demonstrated that certain fine fibres ramifying in the sheath of the nerve are not connective tissue, as usually stated, but true nerve-fibres, which are nearer to their ultimate ramification than the dark-bordered fibres with which they ramify.

3. That the arrangement of the fine nerve-fibres in the form of networks or plexuses upon the surface of the muscle, beneath the skin, and in other parts, resembles that of the dark-bordered fibres in the same situations which has long been known. In fact the networks and plexuses of fine pale nucleated nerve-fibres in the embryo which become the networks and plexuses of the dark-bordered fibres of the fully developed animal, resemble in all particulars the networks and plexuses of fine fibres described by me in the latter, and that in all cases where a trunk composed of fine fibres or coarse fibres branches off from another trunk, the former invariably consists of fibres pursuing opposite directions, as shown in the figure.



4. That by independent observations upon the bladder, small arteries, the skin, connective tissue, cornea, papillæ of the tongue, and other tissues of the frog, I have proved that networks, the fine fibres entering into the formation of which are compound, or plexuses of very fine nerve-fibres, exist, and that nerves do not terminate by free extremities in any of these tissues.

5. That a number of observations I have made upon the minute arrangement of nerve-fibres in the trunks of nerves and upon the origin of nerves from nervous centres in numerous vertebrate and invertebrate animals, favour the conclusion that in all cases complete nerve circuits exist, while during many years of careful observation I have not been able to discover one fact which is incompatible with such a view.

6. That by direct observation upon the thin mylohyoid muscle of the frog, finer fibres than have been hitherto described have been demonstrated, and that some of these have been followed from one compound nerve-trunk over a great many elementary fibres to another trunk. Many of the nerve-fibres described by me in my recent memoirs are so very fine, that it is not surprising that they should have been regarded by many observers as delicate fibres of fibrous tissue. In my preparations their true nature is however demonstrated by the change

induced by the action of acetic acid, and by their continuity with true dark-bordered nerve-fibres.

7. That in the auricle of the frog's heart and also amongst the elementary muscular fibres of the frog's tongue very fine nerve-fibres, not more than the 1-60,000th of an inch in diameter, have been traced from one nerve-trunk to another.

8. That similar facts have been observed in the breast-muscle of the frog, and fine fibres have been traced from one trunk to another.

9. That from all this evidence the conclusion is, that nerve-fibres never end or terminate by free extremities, but that in all cases complete circuits exist, and the inference is justifiable that a circuit is the fundamental arrangement of a nervous apparatus.

A CONTRIBUTION TO THE ETIOLOGY OF TYPHOID FEVER— A RECENT OUTBREAK AT CHARMOUTH, DORSETSHIRE.

By W. B. Kesteven, F.R.C.S.

The village of Charmouth, about two miles to the east of Lyme Regis, consists of a single street running nearly east and west up a steep hill, parallel with the coast line. It is ordinarily one of the healthiest villages in England, enjoying the advantages of an abundant water supply, and the purest of air, both from the sea and beautiful country inland. The houses are, for the most part, detached, or semi-detached villas and cottages, on each side of the main street, which has neither back streets nor crowded courts running off from it. At the foot of a hill a small river, celebrated for its trout fishing, finds its way through pleasant meadows to the sea.

Charmouth is situated on the Lias beds, which offer great attractions to geologists. These strata dip towards the coast at a moderate angle to the horizon and form usually a reservoir, whence issues through countless land-springs an abundant supply of clear and wholesome water.

Thus, under ordinary meteorological conditions, this village possesses all the natural advantages essential to public health, and thus its reputation in all sanitary respects has stood deservedly high. These natural advantages had been still further enhanced by the construction, a few years back, of a sewer down the street, emptying its contents into the stream that passes beneath a bridge at the foot of the village, towards the sea.

During the last summer, however, the long continued

drought had changed this favourable state of things, assisted, as it seems to me, by one fatal oversight on the part of the inhabitants of Charmouth. It has been stated that water flows abundantly by land-springs from the surface of the soil. These may be seen in many of the gardens in front of the cottages that line the road side. The water that is not used is allowed to run to waste by any channels it may find into the fields or brooks. Having observed the early course taken by the fever in this village, I am disposed to attribute its first appearance entirely to sewerage emanations arising out of this waste of water that should be used for flushing. The first cases occurred at the lowest level, the disease gradually creeping up the hill. This would be the result of the retarded flow of the drainage, and consequent accumulation of sewerage at the lower end of the drain. I had taken a house at this part of Charmouth for six weeks, on account of previous ill health in my family. On the day before our return to London one of my children, a boy of eleven years of age, was ill, and in the course of two or three days afterwards two younger children followed suit. All had enteric fever, with the well-marked typhoid eruption; all suffered severely and were not convalescent in less than six or seven weeks. No other cases occurred in our household after our return; but I am informed by my friend Mr. Norris, of Charmouth, that he has had many cases of typhoid since; that four cases proved fatal; and that in almost every case the disease could be traced to bad drainage.

There existed, however, in Charmouth, as in most other country villages, a further possible mode of the propagation of the fever, which would explain its occurrence in several members of one household, more closely than by supposing contagion, the communication by which is not the characteristic of typhoid, as it is of typhus. The water supply of most of the houses is got from wells in the gardens, which, owing to the geological structure of the country, need not to be dug deep before, in ordinary seasons, a copious flow of water follows. During the past autumn these wells were, many of them, dried up, or ran very short of water. Not far from the wells there would be found, in most instances, a privy with its cesspool. It is clear that with such a geological structure as exists in Lias strata the diminution of the water from the wells would induce a percolation of the fluid contents of the adjacent cesspools. This would not be indicated either in the taste or smell of the drinking water, as it is well known that water loaded with the deleterious nitrates, under such circumstances, has rather a pleasant flavour.

I believe that such was the mode of conveyance of the poison, in some cases, but it should be added that when the water in the wells became very scarce, the cottagers at the lowest part of the village were driven to take their supply from the river, into which the drain emptied itself, and thus, furthermore, the disease may have had its origin in some instances.

In the history of this outbreak we have additional evidence that typhoid fever may be developed, from the emanations of sewers. These conditions, it is true, being, in the instance now related, beyond the control of man, viz., the excessive heat and long drought; although it remains very probable that had the means so abundantly at hand been made use of for flushing the drains, the conditions which gave rise to the epidemic would not have been produced. Had the surplus water been conducted by a few pipes into the sewer, the waste water would have done good service in flushing the drains, and probably would have averted an outbreak of typhoid that occurred during the past autumn, the village having been previously free, and, indeed, generally exempt from fever. If this view be correct it follows that the inhabitants of Charmouth have in their own hands the power to prevent the recurrence of such an outbreak in their delightful and otherwise most salubrious village.

This circumscribed outbreak bears out exactly the received doctrines on the non-contagious nature of enteric fever. No case had occurred in this village for upwards of seven years, and on that occasion the disease had been traced to defective drainage. The cases that occurred were all typhoid, or enteric, they were met with in the better class of houses as frequently as among the poor; in short this outbreak was but a repetition of what has been recorded of other limited epidemics, that where more than one person in a house were attacked, all had been alike exposed to the sewage emanations; that it was not traceable in any degree to overcrowding, and that if transmitted by other means than the poisoned atmosphere from the drains, it was conveyed by the contamination of the water supply from the infiltration of sewage from the previously infected.

ON THE OCCURRENCE, DIFFUSION, AND ACTION OF THE CONSTITUENTS OF THE BILE IN THE ANIMAL AND VEGETABLE ORGANISM.

By Dr. F. W. Beneke, Acad. Teacher in Marburg, Giessen, 1862.

(Abstract by Dr. Duffin, Asst. Physician, King's College Hospital.)

In the sixth volume of his Archives (1854), Virchow published a paper "On the Extended Distribution of a Substance Analogous to Animal Nervous Matter." This substance he named myelin (markstoff), suggesting that even should it not ultimately prove a simple body, its occurrence in a uniform thoroughly homogeneous state as much vindicated its claim to a distinctive title as is the case with albumen, syntonin, fibrin, etc., which are also probably compounds.

Virchow describes this substance as characterised by its peculiar brilliancy, and by the strange forms that it assumes. "At one time I saw long threads resembling large primitive nerve-fibres in breadth and shape, extending across the field of vision. They generally exhibited a fine clear axis, readily comparable to an axis cylinder, and a broad, double, definite outline, the outer of which was darker than the inner. They either terminated in a rounded definite margin, or they formed a knot of closely coiled contorted bands, often of irregular thickness, from which again large drops with a double outline sometimes issued. In other situations large, rounded, concentrically striated bodies appeared, suggesting that one of the threads just described had been coiled round itself. This supposition was the more likely, as often processes shot out from these coils that were themselves again coiled at their extremities. At other points the processes ended in small rounded or oval drops, still retaining the double outline. Other masses presented a dull, homogeneous, or partially plaited aspect, round the margin of which the double line everywhere was to be traced. From these double contours, varicose threads shot out in many directions, ending in the most variously twisted and knotted masses. Then some curious things were seen, resembling rolled up pieces of paper, doubly contoured, and included in crooked, somewhat undulating lines. Lastly, smaller forms were very frequent, simple, not doubly contoured, pale, resplendent drops, of the size of a blood corpuscle and less; longer, doubly contoured drops, either spherical or prolonged into a small, short thread;

smaller, shorter threads, with a double outline and perfect termination, often with a doubly contoured drop resting upon them, so as to closely resemble fungi. All these formations consisted of a viscid material, the flowing of which could be often observed as the long, nerve-like threads issued from the general more coherent mass."

Virchow first obtained this substance from diseased pulmonary tissue, but he shortly afterwards procured it from the gall-bladder, a hepatic cyst and the ovary of the calf. He was next able to verify its abundant existence in every spleen, and he also extracted it from the thyroid, from the medullary part of the brain, from fresh pus, and from fibrin. He filtered a hot alcoholic extract, and brought the pellicle which formed on the filtrate with some fresh alcohol into the field of the microscope. Experience seems to leave little doubt that it occurs in the interior of the tissues themselves, and as we may conclude that it is constantly present in small quantities, even in the blood itself, it would not be too much to assume that it is one of the most diffused principles of the animal economy. Of its chemical properties Virchow thus writes, "Easily soluble in hot alcohol, a part only deposits on cooling. In water the mass swells up to an extraordinary degree. It is when thus swollen that it presents its morphological peculiarities. Ether, chloroform, and turpentine readily dissolve it; weak acids and alkalis have little influence; strong alkalis cause it to shrink; strong acids make it swell still further, and then destroy it; chromic acid renders it hard yellow; strong sulphuric acid colours it red, occasionally violet." (Op. cit. p. 566.)

From the time of the publication of Virchow's paper, myelin seems to have been lost sight of, till Mettenheim found it in the human lens. The only addition that he, however, made to our acquaintance with it was the discovery of its polarising properties whilst in alcoholic solution, properties of which water deprived it. The earlier notices as to the nature of myelin are peculiarly scanty. Reasoning from some ultimate analyses, Virchow observes, "From its proportion of nitrogen and phosphorus, from its swelling in water, its behaviour with potash, its combination with lime, we are strikingly reminded of Frémy's cerebric, and oleophosphoric acids." Kölliker* also discovered a substance analogous to these same acids in the semen of the carp, and was struck with its resemblance to the myelin of Virchow, but did not pursue the subject. Little indeed seems to have been done to establish either the chemical constitution

* Zeitschrift für wissenschaftl: Zoologie, p. 257.

or the physiological properties of this substance till Prof. Beneke was gradually led to adopt the views it is the object of this paper to enunciate.

Whilst experimenting with concentrated sulphuric acid on a variety of tissues, he noticed the coloration taken up by the nuclei of some and the fluids surrounding others to be reddish, yellowish, or violet; thus the nuclei of the cornea and the fluid surrounding the lens of the calf acquired the latter tint, whilst the epithelium of the skin of the fœtus underwent a regular play of colours, yellow, reddish, carmine. With an epithelial cancer the play of colours was so marked as to suggest the presumption of nerve fats, and the possibility of cholesterine or myelin, and Beneke particularly noticed the coloration to be most intense about isolated fat drops. The surrounding fluid of the preparations also gradually got tinted, suggesting that the body causing the colour was soluble in sulphuric acid. This last fact led Beneke to suspect the presence of the biliary acids. He was thus induced to test microscopical preparations with a sugar solution (1 sugar to 10 water, in the later experiments 1 sugar to 4 water), followed by a drop of concentrated sulphuric acid. He first tried a cancroid of the penis. A play of colours ensued through yellow, red, carmine to violet, and was most intense at the nests of the cancroid, at the large nuclei of the cancroid cells, and on a few isolated fatty particles. He then exposed with very similar results the tendo achillis of the calf, the cornea of the calf, and permanent cartilage. The cartilage cells especially acquired a deep coloration. The lacunæ of bone became deeply tinted. In from five to ten minutes fat globules would assume a deep rose or violet hue, etc. The presumption was thus raised that all the tissues contained a material yielding to Pettenkofer's test the reaction of the biliary acids. Struck with the vividness of the tints afforded by both normal and diseased epithelial structures in rapid growth, in multiplying cartilage cells, and in the nervous centres, he concluded that the material in question was of great importance in cell formation. This led him to prove the behaviour of yolk of egg, where he found the reaction in great splendour. Immediately on the addition of the reagent a red colour passing into violet was developed, and at the end of six hours this had become deep violet.

These observations he supplemented by the inquiry, do only the biliary acids, or do other constituents of the animal organism yield a reaction with sulphuric acid and sugar, such as Pettenkofer has described for the former?*

* The following are Pettenkofer's directions :—"Pour a portion of the sus-

Butyric, baldrianic, caproic, caprylic, and caprinic acids all gave positive results to the colour test, but never with great intensity. The reaction of lactic acid was so indefinite, that it may practically be considered as insensible to the test. Oleic acid on the contrary gave the nearest approach to choleic acid of any animal substance. Stearic acid and stearin gave very imperfect results, margaric acid and margarin gave none. The conjugate acids glycocholic and taurocholic behave just like their non-nitrogenous constituent cholic acid. This and not the nitrogen holding glycin, or the sulphur holding taurin gives the reaction. Choloidic acid behaves similarly. The reaction of cholesterine varies with the degree of concentration of the sulphuric acid. With certain dilutions a play of colours is indeed obtained, but this is always very transient, and passes into a dirty green. Under the microscope the edges of the crystals become successively yellow, yellowish brown, yellowish red. At last the crystals melt away, and come to resemble a fluid fat. All the ordinary animal albumens must be thoroughly purified from fat, but pulverized lens, the albumen of bullock's blood, and fibrin nevertheless exhibited Pettenkofer's reaction, although less forcibly than before the extraction of the fats. This led Beneke to ask whether the albuminates may not be considered conjugate substances, one of the factors being non-nitrogenised, and acting the same part as the cholic acid of the tauro and glycocholic acids? The idea is supported by the absence of even a trace of reaction between sugar and sulphuric acid, and all exactly known nitrogenised bodies, as leucin, tyrosin, taurin,

pected fluid into a test tube, and add English sulphuric acid, guttatim, to about $\frac{2}{3}$ the volume of the fluid, whereby the temperature is considerably raised. The addition must be made so gradually that the temperature shall at no time exceed 50° R., as otherwise the choleic acid is too much changed. Then add 2—5 drops of ordinary cane sugar solution, containing 1 part sugar to 4—5 parts of water, and shake the whole. If choleic acid be present, a more or less deep violet red colour will be produced, according to the amount of bile present."

Neukomm (ueber die Nachweisung der Gallensäuren, &c., 1860) proposes the following modification (p. 107):—"A single drop of a $\frac{1}{20}$ per cent. solution of cholic or glycocholic acid will yield a splendid purple violet colour if it is brought in contact with a drop of dilute sulphuric acid (4 water 1 sulphuric acid) and a trace of sugar solution in a porcelain cup, and then gently warmed over a spirit lamp. As 1 cubic centimetre equals about 8 drops, it is thus possible to demonstrate $\frac{6}{100}$ milligr. of biliary acid with complete accuracy." As a further test, he suggests (p. 117) "The biliary acid or salt is to be sprinkled with a small quantity of concentrated sulphuric acid, moderately warmed, and then water added. The resinous flocculi that subside are to be separated from the acid, washed with water, but not so as to remove all the sulphuric acid, and then again gently heated in a porcelain cup till coloration ensues. If the residue be taken up in a small quantity of alcohol, and the green solution be evaporated, the interior of the cup will be coated with a deep indigo-blue film, even when but little acid has been used. If the biliary acids have been impure, or the sulphuric acid or the temperature react too long, the pigment film will be green."

urea, etc. Hence all the fatty acids of the body, but particularly the oleic, react in some measure to Pettenkofer's test. The albuminates do the same, and the more markedly, the less completely they have been freed from fats. This test consequently does not enable us to draw any absolute conclusion as to the presence of a biliary acid; but inasmuch as the proper play of colours with the biliary acids is peculiarly characterised by clearness and intensity, and the purple-violet tint is retained for a considerable time, these signs are strongly suggestive of the presence of cholic acid or its compounds, and the test is still possessed of great histo-chemical value.

These results led Beneke to apply Pettenkofer's test to the alcoholic, ethereal and chloroform extracts of a variety of tissues. An alcoholic extract of the yolk of egg gave results as splendid as those obtained from pure cholic acid. But the oleic and other fatty acids which are contained in the yolk of egg are with difficulty taken up by alcohol, albuminous compounds even less; thus the idea of the biliary acids or of their derivatives was again suggested. The alcoholic extract of a number of other tissues, *e.g.*, cartilage, muscle, brain, lens, also gave more or less marked results to sugar and sulphuric acid. The ethereal extracts required to be first dried and taken up by alcohol, as the ether itself, however pure, tends to give the reaction in question, but with this precaution the results were the same as above. The chloroform extracts also gave the characteristic play of colours, but much less vividly. The extracted albuminous tissues, as might have been anticipated, continued to answer to the test. It matters not, however, how in the latter case the reaction is set up; it is certainly not produced by an albuminate in the former, whilst its intensity suggests the presence of biliary acids.

The other properties of the cold alcoholic extracts of animal tissues proved also very instructive. They had all a more or less fatty character, and eagerly absorbed water. All burning with a distinct acrolein odour, they, for the most part, emitted no smell of burning nitrogenised matter, and left a small phosphatic residue. Some of the residues, particularly those from cerebral tissue and yolk of egg, contained crystals of cholesterine. All, when treated with water, swelled up to form a tough, viscid mass. With excess of water a great part of the majority of them dissolved; sometimes the whole. When this process of tumefaction was watched under the microscope, the forms ascribed by Virchow to myelin were severally observed—gradually vanishing as the substance dissolved in the water. Weak alkalis assisted the development of the forms, whilst the

acetic and mineral acids checked it; concentrated sulphuric acid rapidly dissolved them, at the same time inducing a faint violet coloration. These results were obtained with alcoholic extracts of the contents of the jejunum of a dog that had been fed five hours previously on fat beef, from the chyleducts of the mesentery of the same dog, as also from its spleen, venous, and arterial blood. He also got them with the purest fibrin that can be obtained, and from dried blood albumen, from yolk of egg, and from a long list of healthy and diseased structures.

Proceeding to a closer analysis of the myelin thus frequently obtained, Beneke started from the distinct acrolein smell it emitted on burning, the intense reaction of biliary acids it evinced to sugar and sulphuric acid, and its solubility in water. This last peculiarity was strongly opposed to its being any ordinary fat, whilst the first was equally presumptive of the presence of one or more glycerine fats, or of the glycerine phosphoric acid discovered by Gobley in the yolk of egg. Beneke employed the yolk of hen's eggs in his further investigations. He saponified with potash, and boiled the mixture from 1½–18 hours. A clear, golden yellow viscid mass resulted on cooling, in itself almost conclusive of the presence of a glycerine fat. On decomposing with hydrochloric acid, and collecting the fatty acids on a filter, the evaporated filtrate developed when burnt the acrolein odour; but this also occurred with the mass on the filter, which, moreover, yielded the biliary acid re-action more brilliantly than before saponification.

After the mass on the filter had been taken up by hot alcohol, and the latter had been expelled, the residue proved very hygrometric, and developed the myelin forms on the addition of a potash solution. If the alcohol were slowly evaporated, crystals developing the myelin forms first appeared; then crystals of margaric acid; lastly, a volatile fatty acid, probably oleic.

Did the mixture of substances composing the myelin of Virchow contain nitrogen? As already observed, the alcoholic extracts of brain, egg yolk, etc., when incinerated, emitted an odour of burning albuminates. Frémy had already produced a nitrogenized acid, the cerebric, from brain extracts, and Beneke is now prepared to assert the presence of nitrogen in the extracts of egg yolk and of muscle, but the nitrogenized constituents are in extremely small quantity.

The further processes were conducted as follows:—The alcoholic extracts of a number of yolks were dissolved in water, and converted into a lead soap by means of litharge. After decantation the lead precipitate was extracted with hot alcohol,

filtered, and the extract cleared of lead by sulphuretted hydrogen. On cooling, a myelin free of any of the ordinary saponifiable fats was obtained. The shapes of these crystals resembled those ascribed by Virchow to a special form of cholesterine, but they yielded the cholic acid reaction perfectly, and their great solubility in water was opposed to their being cholesterine; still cholesterine they proved to contain. As this substance is almost insoluble in cold alcohol, this was used to wash the mass, which was then recrystallised out of hot alcohol as snow white silky crystals. These, on ultimate analysis, gave the following results:—

HO	= 0·2195	= 11·98 H*
CO_2	= 0·6255	= 84·01 C
O		= 4·01 O
		100·

But what was the residual substance that imported solubility in water to cholesterine and caused it to assume the shapes of myelin? The intense reaction with sugar and sulphuric acid pointed strongly to the biliary acids. A myelin yielding extract was examined for chromogens according to the methods of Frerichs and Staedeler (see ante, note, p. 195). The honey-like, golden yellow extract was dissolved in concentrated sulphuric acid. A viscid solution, with a carmine red edge, was thus obtained. On the addition of cold water, the reddish tint vanished, and greyish flocculi fell. These being dissolved in fresh alcohol yielded to sugar and sulphuric acid a distinct reaction of cholic acid. When these flocculi were warmed with a little spirit in a porcelain cup, the vessel became coated with a blue, slightly reddish covering. The next day this covering was green.

The alcoholic extracts of the lead soap were then evaporated to two-thirds their volume. Allowed to crystallize in the cold, they precipitated fresh quantities of cholesterine. These were filtered off, the filtrate again reduced, and crystallized out, and so forth, till only yellowish flocculi appeared in the deep golden yellow mother-liquor. Thus a mother-liquor was obtained, from which the greater portion of the cholesterine had been removed, but which must still contain any biliary acids present. This mother-liquor, when treated with sugar and sulphuric acid, yielded the most intense reaction of biliary acid, but on evaporation no crystalline residue was found, but a tough,

* Schwendler and Meissner give the percentage of cholesterine as C = 84·2, + H = 12, O — O = 3·8 — 100.

golden yellow, hygroscopic mass, containing scarcely a particle of crystalline cholesterine. The mass dissolved easily in water, but, curiously enough, when examined with the microscope, none of the characteristic beautiful shapes of myelin were perceived, but only large globular masses without the remotest resemblance to it; but the addition of a fresh portion of cholesterine at once reproduced the form. Hence the presence of cholesterine is absolutely necessary for the production of myelin. But what was the fatty viscid body, soluble in water that had been now quite deprived of cholesterine? It had a neutral reaction, and when burnt yielded a feeble but distinct odour of acrolein. The suspicion of the presence of an oxide of lipyl combined with a biliary acid was thus raised. Beneke first essayed to obtain the biliary acids by prolonged boiling with baryta. To deprive the mother-liquor of any trace of cholesterine, the alcohol was expelled, and the residue dissolved in water; the solution was then boiled for ten hours with baryta water; it then reddened and deposited a resinous precipitate. The liquor, after filtration, was exactly neutralized with sulphuric acid, again filtered, and evaporated. A tough, brownish residue was thus obtained, containing a number of crystalline needles. The whole residue showed very little solubility in alcohol, and gave so slight a reaction with sugar and sulphuric acid as to banish the idea of the presence of cholic acid, but the residue, for the most part, dissolved easily in water, and then alcohol precipitated a yellowish tough mass, whilst alcohol acidified with hydrochloric acid, threw down a scanty whitish precipitate, consisting of the aforesaid needles.

The baryta precipitate itself, on the contrary, continued to react vividly to sugar and sulphuric acid. After the withdrawal of the baryta by sulphuric acid, it deposited first a trace of cholesterine, and later a tough, yellow, fatty body, identical with that acquired by direct evaporation of the mother-liquor. The same gave the biliary acid reaction still most intensely, but it had quite lost its solubility in water. When mixed with cholesterine and brought into contact with water, it refused to assume the forms of myelin, but the addition of alcohol still produced them.

These results would be inexplicable but for the researches of Kolbe* into the behaviour of taurin and glycin. Boiling with baryta separates the biliary acids into cholic acid, and its nitrogenized conjugates taurin and glycin. Both of these latter are soluble in water, insoluble in alcohol. Whilst glycin can com-

* Ueber die chemische Constitution u. Künstliche Bildung des Taurin. Annal. d Chem. u. Pharm. cxxii., Hft. i, p. 33.

bine both with bases and acids, taurin has no basic properties, but can unite with bases. Further, taurin remains in solution in alcohol containing some alkali, but is readily deposited from acidified alcohol, whereas glycin is readily precipitated from its watery and alkaline solutions by alcohol, but in an acid form is readily kept dissolved by alcohol.

Assuming, then, that the fat-like soluble body of the mother-liquor contained taurochlorate and glycochlolate of lipyl, it is not improbable that by boiling with baryta taurin and glycin may be retained in solution as taurinbaryta and glycinbaryta. Now the baryta was removed by sulphuric acid. When the residual solution was alkalized and treated with excess of absolute alcohol, a tough brownish mass separated, consisting essentially of glycin; and on the alcohol being acidified, a light whitish crystalline precipitate fell, probably taurin.

Beneke concludes that the body which was insoluble in water was cholate of lipyl, as it continued to yield the biliary reaction, and on the addition of cholesterine and alcohol developed myelin forms, whereas the glyco- and taurocholates developed the same properties to water or thin syrup.

Thus to sum up. There are in the alcoholic extract of the lead soap, firstly, cholesterine. When this has been as far as possible crystallised out, an unsaponified fatty body remains in the mother-liquor, from which boiling with baryta extracts, taurin and glycin (?), but leaves an unsaponified fatty body, giving an intense biliary reaction to sugar and sulphuric acid, and which Beneke takes to be cholate of lipyl.

All attempts to trace the biliary acids in healthy blood and tissues have, as is well known, hitherto been in vain; yet since the researches of Bidder and Schmidt it is scarcely doubtful that they re-enter the blood either directly or with the chyle. Is it then very unlikely that the constituents of the bile combine with the food whilst in the intestine, but that this combination has hitherto eluded us in the blood and the tissues? From the first Beneke considered it probable that the oxide of lipyl, when separated from its fatty acids, might combine with the biliary acids, and so pass into the blood, and that possibly a combination of cholesterin with the fatty acids might take place. These presumptions are countenanced by the following considerations:—

I. Bernard has indicated the faculty possessed by the pancreatic juice of separating the oxide of lipyl from its fatty acids, and although Lenz* correctly remarks that the acid of the gastric juice can arrest this action, still he further

* De adipis concoctione et absorptione. Dorpati, 1860. Page 61.

observes "hic effectus succi gastrici, quo succi pancreatici actio in dissolvendos adipes turbatur, si acidum ejus sive natro, quod in bile inest, sive diversa via addito kali caustico neutrale redditur, r u r s u s t o l l i p o t e s t." As the chyme traverses the intestine, the gastric acid is gradually neutralized, and the pancreatic juice is then free to decompose the neutral fats. This gradual neutralization of the gastric acid has also its peculiar advantage, as it allows fresh and fresh portions of oxide of lipyl to be presented in *statu nascenti* to the biliary acids, in which state only combination appears to ensue. Thus it is provided that free biliary acid, pancreatic juice, and neutral fat should first be thoroughly intermingled, and only when enough bile has been secreted to completely neutralize the gastric acid does the influence of the pancreatic juice on the fats come into operation, followed by the immediate union of the oxide of lipyl with the biliary acids.

II. It is manifest that especial accessories are requisite to carry the fats through the watery blood fluid, as also to conduct them again out of the vascular system. It is further *à priori* probable that the bile presents the necessary requisites for these purposes, and that it not only itself passes in a soluble form into the blood, but imparts solubility in water to the fats and fatty acids. It is, indeed, true that a certain amount of fat is absorbed even when no bile reaches the intestine, but this is quite intelligible, as Beneke has found myelin, and at least one biliary constituent, in all parts of the animal organism, and even in the vegetable kingdom (*ut infra*).

III. The access of the bile only allows the absorption of a definite quantity of fat in each species of animal.

IV. Experiments on animals with biliary fistulæ show with certainty the importance of the bile, not only in the absorption of the fats, but for the general economy of the individual. All animals deprived of their bile could only be kept alive for a short period by the supply of enormous amounts of food, and all ultimately died of total inanition. If, now, a biliary constituent be essential to the production of myelin, and myelin be an essential component of nervous matter, this result is quite accounted for. The animals survive so long as the amount of myelin present in their food scantily suffices to supply that consumed in the organism. If these considerations be correct, myelin ought to be extracted largely by means of cold or hot alcohol from the contents of the intestine. This was actually the case. The meat supplied to the animals that were the subjects of experiment contained small quantities of myelin.

The extract prepared from the contents of the duodenum above the bile duct yielded but a trace of it, but the contents of the jejunum presented it in enormous quantities. Similar quantities were also obtained from the chyle.

It thus became of great moment to ascertain the relations of the biliary acids to glycerine. Both cholic and glycocholic acids were found to dissolve freely in glycerine, but the solutions had none of the characteristics of myelin. When treated with dilute mineral acids they became turbid, but only developed minute spherules. Almond oil, treated similarly, at first seemed to give an affirmative result, but subsequently was found unfit for experiment from the presence of preformed myelin; indeed, the difficulty of obtaining a fat quite free from this substance was for a time insuperable, till at last a pure stearin was prepared by repeated extraction with ether.

With this stearin the act of digestion was as nearly imitated as possible. To stearin kept at a temperature of 70° C. a small quantity of dilute hydrochloric acid was added, then quite fresh ox gall, then freshly expressed pancreatic juice, the whole being gradually neutralized by excess of potash solution. At the end of an hour, phosphoric acid was added in excess, and the mixture cooled. The stearic acid and a mass of whitish flocculi gradually rose to the surface. These were collected and treated for twelve hours with cold alcohol, then filtered and evaporated. A yellowish mass remained, which developed the myelin forms under the microscope. Ox gall was used in these experiments instead of the biliary acids, as experience had already pointed out cholesterine as a constant component of myelin. To at once obtain the right proportions of cholesterine and biliary acid it was thought advisable to resort to the ox gall. As initial experiments, the absence of myelin from ox gall was demonstrated, as well as its absence from a mixture of cholesterine and biliary acid. Thus the only conclusions that remain are either that the biliary acids unite with oxide of lipyl in *statu nascenti*, and that this combination, mixed mechanically with cholesterine, develops the forms of myelin, or that simultaneously with the union of the oxide of lipyl to the biliary acids, a combination of the liberated fatty acids with the cholesterine of the bile ensues, and so these forms are generated. The latter hypothesis Beneke regards as most unlikely. Even after ten hours' boiling of the cholesterine mother-liquor of the egg yolk with potash in alcoholic solution, no decomposition resulted. Thus the artificial production of myelin confirmed the view that it consisted of a combined biliary acid with oxide of lipyl mixed with cholesterine. It was, moreover, impossible to reproduce

the pure biliary acids from this combination just as they had failed to be obtained from the naturally produced myelin. It may well be asked whether this combination may not behave differently to other combinations of the oxide of lipyl, and may not be intended to enter into new associations in the economy, but never again to decompose into glycerine and biliary acid. In proportion to the amount of cholesterine added to the artificial myelin were the general characters of the shapes produced. The more cholesterine is added, the tougher and more resinous does the mass become, and simultaneously the microscopic forms increase in tenuity.

In a later series of investigations Prof. Beneke was enabled to obtain myelin from a number of products of the vegetable kingdom. Thus fine olive oil yielded it on boiling with potash, and extracting with alcohol. The young parts of a number of plants, such as the green leaves of fumaria, young asparagus, potato germs, the pollen grains of fumaria, and of lonicera tartarica alba also yielded it. It was also found pre-existing in great abundance in the ordinary pisum sativum. Simple extraction with cold alcohol for twenty-four hours produced it largely. The seeds of a great number of plants also yielded it, as of the white bean, rye, wheat, rape-seed, etc. Lastly, he obtained cholesterine from the pea and from olive oil.

ON MUSCULAR CONTRACTION.

By H. F. Baxter, M.R.C.S.

As the present inquiry must be considered a continuation of my previous investigations, I will briefly recapitulate some of the conclusions, partly deduced as corollaries from other conclusions, which I have been led to entertain in regard to muscular contraction. In a paper* "On the Polarized Condition of the Muscular and Nervous Tissues," it was stated, "During life, the muscular and nerve fibre may be considered as existing in a state of t e n s i o n, a f o r c e d state, and muscular relaxation may coincide, and would be synonymous, with this polarized condition or state of t e n s i o n and contraction, the result of a depolarization of the muscular fibre. In the normal state this d e p o l a r i z a t i o n is induced by means of

* Edinburgh New Philosophical Journal. April, 1858. Essay on Organic Polarity, chapter ix.

nervous agency; but it may also occur from other means, such as chemical or mechanical agencies, or whatever is likely to produce disorganization. Depolarization having occurred, the polarized state is easily and readily restored by the act of nutrition; hence contraction may be partly considered as the result of molecular attraction. The particles constituting the muscular fibre being in a state of self-repulsion, whilst in this polarized condition, would resist the force of molecular attraction unless depolarized; and, according to this view, the results of muscular contraction would necessarily follow, in conformity to the law as laid down by Schwann." In a subsequent investigation "on Muscular Power,"* I was forcibly struck with the importance of an elongated state of the muscle prior to contraction, the contractile power of a muscle increasing, up to a certain point, as the elongation of the muscle was increased.

The views that are at present entertained in regard to the influence of nervous agency over muscular contraction renders the question of muscular contractility an open one, and although my own opinion coincides with those who consider contractility as a property of the muscular fibre, independent of the nerve, still as the opposite opinion, that it is dependent upon nervous agency, is entertained by eminent physiologists, especially amongst the Germans, it behoves us to examine into the grounds upon which this conclusion is based. In discussing the question of muscular contraction it will, however, be necessary to bear in mind the two questions that are involved—1st, the contractility of the muscular fibre; and 2ndly, the contraction induced by nervous agency. The terms contractility and contraction, when employed to designate the same idea, appear to me to have created a great deal of confusion in the discussion of this subject; muscular contractility need not necessarily imply nervous agency, but muscular contraction may do so.

The greatest difficulty in solving these questions arises at the very commencement of our investigations, namely, the ignorance we are in as to the mode in which the muscular fibres are supplied with nerves. Microscopical anatomy has not yet decided this point for us; we only know that muscular fibres are freely supplied with nerves, and consequently the impossibility of deciding how far the means which we employ, whether chemical, mechanical, or electrical, for the purpose of exciting contractions, act first upon the nerves, or upon the muscular tissue alone, invalidates, to a great extent, any conclusion that

* Edinburgh New Philosophical Journal. October, 1863.

may be arrived at by their employment. For instance, if we use chemical or electrical means, the stimulating effect is not confined to the spot, but extends more or less over the surface of the muscle, and although Kuhne* has shown that a difference is observed in the effect produced by chemical agents, when applied to the trunk of the nerve or to the muscle, still the objection may be raised as to their limited action upon the muscular tissue. Upon these grounds I consider that the excitement of the muscle by mechanical means, such as the point of a needle or a piece of glass, would give more trustworthy results, but even here the same objections may be raised under the supposition that the surface of the muscle at the point of irritation may be connected with nervous tissue.†

If we could destroy the action of the nerve by paralysing it to its extreme ends without injuring the muscular tissue, a great point would be gained. It has been supposed, and a great many experimental results have been based upon this supposition, that this desirable object can be effected by means of passing an electric current, i n v e r s e l y, along the trunk of the nerve, producing the state which has been designated by Pflüger as a state of anelectrotone. In my present experiments I employed the following method:—

I took the two hind limbs of a middle-sized frog, separated from each other at the pelvis, and carefully dissected out the lumbar nerves down to the sciatic notch. The nerve of the limb *a* was then placed upon two strips of moistened bibulous paper, which rested on two distinct pieces of thick glass, the interval between them being about half an inch. Upon one end of each strip of paper rested the electrodes, which consisted of thick copper wire, and were so bent as to press firmly down upon its surface. The battery consisted of six of Grove's middling-sized cells; the sulphuric acid solution was formed with 1 part of strong acid to 12 of water, pure nitric acid being employed in the platinum cells. The limb *b* with its nerve was arranged exactly in the same manner as that of *a*, but reversed, so that the conditions of the two limbs should be the same. A current was now passed along the nerve *a* inversely, when a contraction occurred at the closing of the circuit in both limbs. The current was allowed to pass for twenty minutes, and during this period the nerves of both limbs were occasionally moistened slightly with distilled water, by means of a camel's hair pencil; an occasional contraction occurred in the limb *a*.

* Report on Muscular Contraction. By A. B. Duffin, M.D. In Beale's Archives of Medicine. Nos. X. and XI. January, April, 1862.

† The researches of Prof. Beale evidently show that the nerves cross the fibre, and form a plexus or net-work *external* to the sarcolemma.

After the passage of the current for twenty minutes, it was intermitted when tetanic contractions ensued in *a*, but upon renewing the current they subsided. I now carefully divided the skin covering the muscles of the thigh and the gastrocnemius in both limbs during the passage of the current, and then irritated the surface of the muscles by means of a pointed needle; the needle being inserted into a piece of glass tubing by means of a cork for a handle. A slight elevation was observed in the muscles of both limbs, at the point of irritation, and which gradually extended in a wave-like manner on each side, in the direction of the muscular fibre. The current was prolonged for thirty minutes, and the irritation repeated with the same results. The current was now suspended entirely, tetanic contractions ensued in the limb *a*, and which lasted, with gradual intermissions, for ten seconds. I now endeavoured to excite contractions by means of the needle, as before, but some time elapsed before any effect could be obtained in the limb *a*, and when it did occur, the swelling was persistent, there was not that wavy motion extending along the muscular fibre, as previously observed; with the limb *b* there was no difficulty in obtaining the contraction. Upon applying a drop of concentrated solution of common salt upon the surface of the muscles, slight fibrillar motions were produced in both limbs, but more extensive in *b*. When the salt was applied to the trunk of the nerves, contraction was produced in *b*, but not in *a*. The current from the battery was now sent along the nerve of limb *a*, direct, but no contraction; but when sent along the nerve of *b*, violent contractions. The current was then sent along the trunk of the nerve *a*, so as to traverse the limb, one electrode being in contact with the tendo Achillis. Slight contractions were now produced upon making and breaking the circuit.

Here, then, we had in these experiments destruction of the conducting power of the trunk of the nerve, still there was slight muscular contractions produced when the muscle was irritated with the point of the needle, and also when the chemical reagent was employed. Of course it will be said that the paralysing influence of the current, the so-called anelectrotonic state of the nerve, was not produced to its extreme ends, either during the passage of the current or the suspension of it; and that the slight effects which occurred when the current was suspended, and after the tetanic contractions had ceased, would show that this was the case. As a different view may be entertained in regard to this point, I will defer stating it until other experiments have been related.

The experiments were repeated on three other limbs in the same manner, but with this difference; in the first the current

was passed for half an hour; in the second for three-quarters of an hour; in the third for one hour. The muscles were irritated during the passage of the current and after the subsidence of the contractions as before. As my object was to obtain, if possible, a complete paralysis of the nerve, it will be unnecessary to enter into a detailed account of the whole experiment. In No. 1, during the passage of the current, there was no difference in the appearance of the muscular contraction when excited in the limbs. After the suspension of the current and when the tetanic contractions had subsided it was more difficult to excite the contraction in *a* than in *b*. In No. 2, during the current, the contraction excited in *a* presented a quivering motion; in *b*, the contraction was more prolonged and of greater extent. After the tetanic contractions had subsided the irritation in *a* produced a decided elevation, which continued for some time and then gradually subsided; in *b* a wavy motion of the fibre was first produced, and then a quivering motion. In No. 3 the irritation produced the same appearance in both limbs, viz., there was first an elevation and then an extended wavy motion on both sides. After the tetanic contractions had subsided in *a*, there was an elevation, and afterwards a slight quivering; whilst in *b* the elevation first arose, and then a wavy motion throughout the whole length of the fibre. The tetanic contractions lasted in No. 1 ten seconds, in No. 2 five seconds, in No. 3 eight seconds. No irritation of the trunk of the nerves, either by chemical or electrical means, produced contractions in *a*, unless the current was made to traverse the entire limb; but contractions were readily excited in *b* by these means.

From these results I can come to no other conclusion than that the passage of an inverse current along the trunk of the nerve is incapable of producing a paralysing effect to its extreme ends, but that a paralysis of its trunk may be induced, and which is dependent upon its disorganizing action, which takes place during its continuance; the disorganizing action, however, does not extend to the extreme ends. The difference in the contractions produced upon irritating the muscle after the subsidence of the tetanizing effects must, therefore, be due to other circumstances.

I now repeated the experiments on three other limbs, employing the current direct instead of inversely. In No. 1 the contractions excited in the two limbs were similar, producing an elevation and a wavy motion extending on both sides. Upon suspending the current there was a single contraction, but no tetanus; the contractions produced by irritating the muscle presented the same character in both limbs, and as

before. In No. 2 a slight quivering, which ultimately terminated in a prolonged wavy motion on each side of the irritated point occurred in *a*; whilst in *b* there was at first some difficulty in exciting contractions, but they then occurred and extended along the fibre. Upon suspending the current a slight quivering occurred in *a*, which soon subsided; upon irritating the muscle the quivering was renewed, then subsided, and a persistent elevation remained; in *b* contraction of the fibre was easily excited, and became more extended. In No. 3 the contractions excited in *a* and *b* were the same, an elevation occurred and then extended on both sides. Upon the suspension of the current there was no tetanus in *a*, but upon irritating the muscle a slight tetanus was produced, which lasted four seconds. Some difficulty occurred in arousing a contraction in *b*, it ultimately occurred, producing an elevation which became persistent.

I was now led, partly from these results and also from the researches of Matteucci in regard to the secondary electromotor power of the nerves, and from my own investigation respecting the so-called electrotonic state of the nerve,* to entertain the opinion that the effects referred to the so-called anelectrotonic state of the nerve, instead of being the result of a paralysis of the nerve extending throughout its whole extent, might be more immediately due to the state of the muscular fibre itself, the fibre, at the time, being in a state either of permanent contraction, or else that the contractile power of the muscular tissue itself had become completely exhausted from previous contractions. Matteucci has established the fact that the muscular current bears some relation to the contractile power of the tissue, and this has been confirmed by some of my own experiments. Here then we have a test which will enable us to judge how far the effects may or may not be referable to a loss in the contractile property of the tissue.

I now repeated the experiments, passing the current along the nerve *a* i n v e r s e l y for half an hour, three-quarters of an hour, and one hour, but in order to prevent the tetanic contractions from taking place upon the suspension of the current, the nerve trunk was divided low down the thigh before the circuit was broken, so as to avoid any contraction excepting that which might occur upon the division of the nerve. A section was then made through the thigh, leaving the lower half, which was separated from the knee joint. The skin was reflected, and thus a single electromoter muscular element was formed; a similar element was formed with the limb *b*, which had been traversed by the d i r e c t current.

* Edinburgh New Philosophical Journal. January, 1864.

In No. 1, when the section was made, there was slight quivering of the muscles in both limbs. They were then formed into a combined circuit with a galvanometer. The current indicated 3°. When each formed a single circuit, the amount was 1°; if anything it was in favour of limb *a*. A differential circuit was now formed; no result upon the needle.

In No. 2 similar quiverings were observed upon making the sections. The combined current was 4°; a single current, that of *a* $1\frac{1}{2}$°, of *b* 2°. The differential current in favour of *b* 1°. This was a larger frog.

In No. 3 similar quiverings were observed, but not so prolonged. Combined current 3°; single current each 2°. Differential current slightly in favour of *b*.

In the following experiments the tetanic contractions were allowed to take place, and the circuits formed, after the contractions had ceased:—

In No. 1 the tetanic contractions lasted 20 seconds. On dividing the muscles they retracted in limb *a*; dividing the muscles of limb *b*, slight quiverings occurred. A circuit was formed with the limb *a*; no effect. With the limb *b* 3°; with the combined current 2°; with the differential current, 2° in favour of *b*.

In No. 2 the tetanic contractions lasted 18 seconds. On dividing the muscles, slight twitchings in those of *a*; in *b* merely retraction of the muscles. The current from *a* amounted to 1°; from *b* 3°. Combined current 3°; differential current 2° in favour of *b*.

In No. 3 the tetanic contractions lasted 10 seconds. On dividing the muscles of *a* an occasional quivering; dividing those of *b* the muscles merely retracted. The current from *a* scarcely affected the needle; from *b* 4°. Combined current 4°; differential current 3° in favour of *b*.

I now passed the current inversely along the nerve *a*, and, before suspending it, divided the nerve, to prevent the tetanic contraction from taking place. When the current was suspended, the muscles were irritated with the point of a needle. Contractions immediately took took place in the limb *a*, and to a greater extent than in those of *b*. When they had subsided, I could easily reproduce them in *a*. In another experiment, after the current had been passed for half an hour, I had some difficulty in exciting the contractions in *b*, whilst in *a* I could easily get them. The limb *b*, instead of being straight, the leg was bent upon the thigh, upon straightening the leg so as to stretch the muscles, and then irritating them, contractions ensued. I now passed the current along the nerve of another frog, and kept the leg flexed upon the thigh.

After dividing the nerve and suspending the current which had been passed for half an hour, slight quiverings of the muscles took place on irritating them. Upon stretching the limb, and then irritating the muscles, the contractions were much more powerful, and more readily obtained. I repeated this experiment, viz., kept the limb flexed during the passage of the current, so as to relax the muscles of the thigh and leg, the flexor muscles. Upon the cessation of the current, stronger and more extensive effects were obtained when the muscles were straightened and elongated by extending the leg than when they were flexed.

These latter experiments beautiful show the necessity of having the muscular fibre elongated, in order to obtain a contraction of the fibre; and the previous experiments indicate the connexion that exists between the electrical state of the tissue, and its contractile power. The electrical state of the muscle does not cause contraction, if anything, prevents it, by keeping the muscular fibre in a state of tension and elongation.

The current was now passed i n v e r s e l y along a nerve for half an hour. The limb was then suspended, and a weight (1 oz.) attached to the tendo Achillis to keep the muscles extended. Slight twitches of the muscles were observed on removing the weight for fifteen seconds; when the contractions had subsided, and then on irritating the muscle with the needle, no effect was observed. The experiment was repeated, and the nerve divided previous to suspending the current, to avoid the action of the nerve. The same weight was used, and after the lapse of fifteen seconds the muscle was irritated, slight quiverings were readily produced. The experiments were repeated, but the weights used were half an ounce and two ounces. The only difference in the results was this: when the nerve was not divided, a slight contraction was produced with the light weight, but none with the heavy weight. When the nerve was divided, contraction of the fibre was easily produced in the limb with the light weight, but with the heavier weight (two ounces) there was a persistent swelling, but no decided contraction.

These experiments show that when the trunk of the nerve which has been traversed by the current is allowed to remain, there is a constant source of excitement in the nerve to cause contraction, and which cannot be prevented by elongating the muscle so that the contraction should not take place. Let the cause of excitement be removed by dividing the trunk of the nerve, and separating it from the limb; the muscular fibre then retains its contractile power. If the weight, however, be too great for the muscle, so as to overstretch the fibre, the con-

tractile power is also destroyed. The question now occurs what effect has this stretching upon the muscular current?

I took the limbs of three frogs separated from the body, and to No. 1 attached 1 oz., to No. 2 $1\frac{1}{2}$ oz., to No. 3 2 oz., and suspended them for half an hour. On comparing the muscular current with those of their fellow-limbs no difference was observed, excepting in No. 3, which was somewhat less than that of the other. These results would appear to contradict the conclusion, that the electric state of the tissue bears a relation to the contractile power of the muscle. We should not, however, be justified in coming to this conclusion, for there is evidently a great difference between the loss of power arising from over-work, from constant contraction, and that arising from over-stretching of the muscular fibre. In the one case, during muscular action force is evolved from internal action; in the other there is no resistance to the extending force; in fact, there is no internal action. The results would be different if the limb was connected with the living animals when a resisting power would be called forth, so as to excite muscular action, as I have ascertained in a former paper.*

An intermittent current was now passed along a nerve for twenty minutes so as to keep the muscles in constant action, in a state of tetanus; on suspending the current and irritating the muscles, slight quiverings occurred. The nerve was divided and separated, the flexor muscles became contracted, and I could not excite contraction in them. I then stretched the limb, and allowed it to remain quiet for a short time—two minutes; upon irritating the muscle an elevation occurred which remained for some minutes; but when the limb was again stretched, I could not reproduce it. The experiment was repeated, but as the results were similar to those observed served when tetanus is produced after the passage of the i n v e r s e current, I need not detail them; there was the same loss in the muscular current as compared to that of the quiescent limb, and the same difficulty in obtaining contractions of the muscular tissue; the contractile power evidently became exhausted. And here I will just observe that the results recorded in the present paper have been obtained upon muscles in limbs separated from the animal, so that muscular action has been attended with a loss of power without the means of restoration, and I would have related some experiments showing the importance of the circulation of the blood in influencing the results, but having already done so in a former paper I must now refer to them.†

* On Muscular Power. Edinburgh New Philosophical Journal. October, 1863.

† Edinburgh New Philosophical Journal. October, 1863.

CASE OF PERFORATING ULCER OF THE DUO-DENUM. GALL STONES.

By Maxwell Mastiss, M.D.

The lady who was the subject of this note had been at various times under my care for symptoms indicative of ovarian disease, and at other times on account of obstinate constipation, vomiting and other evidences of disordered digestion. Active purgation, and enforced abstinence from spirits (to which liquor she was unfortunately far too partial) sufficed to relieve the vomiting. At no time were there any indications of the presence or passage of gall stones. On the 13th April, 1864, after having been for some months in pretty good health, she left home early in the morning, apparently quite well, but about noon, immediately after having taken some soup, was seized with violent pain in the abdomen, and returned to her residence. In the evening I found her writhing in great agony, the pain being referred especially to the umbilical region. Pressure and friction afforded some slight relief. The abdomen was scarcely tympanitic, and not at all tender on pressure. There had been slight vomiting, the bowels had not been relieved for two days, on which account she had taken a dose of castor-oil in the morning. The tongue was moist and covered with a thick white fur. Pulse 70, full and soft. Judging the case to be one of colic, I prescribed a cordial draught containing a little laudanum, ordered the application of a turpentine stupe to the belly, and the use of an enema. The latter brought away a few pieces of flattened fœces of the diameter of a goose quill. A second injection returned unchanged. The pain continued unabated till 4 A.M. the following morning, when a sudden change occurred. I saw her in ten minutes, but she was in a state of collapse, and she died in less than half an hour after the change was first noticed.

Post-Mortem.—Abdomen only examined. General peritonitis, and a large quantity of pus in the cavity of the belly. At the upper part of the duodenum, near the pylorus, was an ulcer of the size of a three-penny piece, the margins of which showed clear evidence of recent perforation, were stained with bile, and had small fragments of gall stone still adhering to them. Lying on the bowel, within an inch from the ulcer, was a black nodular gall stone of the size of a large pea, and much resembling a mulberry calculus in appearance. In the gall bladder itself were a great number of similar concretions, but none in the gall ducts. The intestines were empty and presented no indication of obstruction at any part. The liver and kidneys fatty. Both ovaries were diseased, the cysts were about the size of a large plum, and contained a dark grumous fluid. The peritoneum in their neighbourhood showed many old adhesions.

(*Continued from page* 108.)

Acute Rheumatism.—E. G., æt. 53. Cook. Adm. Mar. 9. Disch. Ap. 22. In H. 44 days. *Recovery.* Dr. Garrod, vol. 1, p. 63. Acute rheumatism 7 years ago. Prev. ill. 3 days. Ankles, knees, wrists, and shoulders affected in succession. Profuse sweating. Basic systolic bruit. Urine albuminous, containing granular casts. On 7th day, friction sound over heart (26 days) pain in. præcordia and dyspnœa. Pains about shoulders till 40th day.

Quinine and bicarbonate of potass (31 *days*). *Then syrup of iodide of iron. Leeches to præcordia.*

Acute Rheumatism.—F. F., æt. 28. Street Keeper. Adm June 9. Disch. June 20. In H. 11 days. *Recovery.* Dr. Garrod, vol. 2, p. 159. Acute rheumatism 9 years ago. Chorea 1 month afterwards. Prev. ill. 1 month. On adm. pain in left shoulder, elbows, and knees. Free sweating. Tongue clean. Systolic bruit over apex of heart. Free from pain on June 18.

Bicarbonate of potass and quinine.

Rheumatism.—Dyspepsia.—M. W., æt. 44. Adm. Mar. 12. Disch. Ap. 1. In H. 19 days. *Recovery.* Dr. Beale, vol. 6, p. 119. Pains in joints, headache, emaciation. loss of appetite. On 11th day after adm. free from pain. Appetite good.

Quinine and bicarbonate of potass. Pepsine and hydrochloric acid.

Subacute Rheumatism.—W. F., 53. Porter. Adm. Mar. 21. Disch. Ap 7. In H. 17 days. *Recovery.* Dr. Beale. Five or six attacks during last 20 years. Prev. ill. 5 wks. Right wrist, left wrist, right knee and ankle, left knee and ankle, affected in succession. Sweating. Tongue slightly furred.

Alkaline mixture, and quinine and opium.

Subacute Rheumatism.—M. O., æt. 16. Servant. Adm. Mar. 17. Disch. May 27. In H. 71 days. *Recovery.* Dr. Garrod, vol. 2, p. 83. First attack. Prev. ill. 1 week. Pain and swelling of right knee. On fourth day pain in left shoulder. Sweating. P. 104. Knee well on 10th day. The shoulder remained painful for some time, and became stiff, so that she could not raise the arm. When discharged she had the full use of it.

Quinine and bicarbonate of potass (16 *days*). *Quinine, sulphate of iron, and potassium and ammonio citrate of iron. Passive motion of left arm.*

Rheumatism.—J. S., æt. 32. Butcher. Adm. Ap. 13. Disch. May 16. In H. 33 days. *Recovery.* Dr. Beale, vol. 6, p. 127. Acute rheumatism 3 months ago. Prev. ill. 6 wks. Commenced in ankles. Then in right knee, wrists, and shoulders. Loss of appetite. Sweating. On adm. ankles and wrists red and swollen, knees swollen and covered with a slight blush. Convalescent about May 5.

Bicarbonate of potass, opium and sulphate of magnesia, with inf. calumbæ. Alkaline lotion to joints. Afterwards blisters above knees, and bicarbonate of potass and liq. cinchonæ.

Rheumatism.—M. L., æt. 22. Servant. Adm. May 26. Disch. June 20. In H. 25 days. *Much relieved.* Dr. Garrod, vol. 1, p. 113. Previous illness 1 month. Pain and swelling of knees. On adm. right knee much swelled, left knee slightly swelled. Tongue clean.

Iodide of potassium. Iodine paint to knees.

Chronic Rheumatism.—J. P., æt. 17. Adm. Feb. 27. Disch. Ap. 22. In H. 54 days. *Relieved.* Dr. Johnson, vol. 16, p. 125. Typhus 10 years ago, followed by deafness. Acute rheumatism 8 months ago; heart not affected. During convalescence pain returned in left knee (3 wks.). Ten weeks ago left hip became painful, and has remained so ever since.

Iodide of potassium and citrate of iron. Blisters over hip. Subdermic injection of morphia. Hot air baths.

Chronic Rheumatism.—A. B., æt. 23. Servant. Adm. Feb. 21. Disch. Ap. 29. In. H. 67 days. *Relieved.* Dr. Johnson, vol. 16, p. 125. Housemaid's knee 2 years ago. Pain in joints and weakness ever since. On adm. wrists and small joints of fingers painful and swollen. Flatulence, loss of appetite, and sour eructations.

Iron and iodide of potassium. Afterwards iron and bicarbonate of potass. Blisters above wrists.

Rheumatoid Arthritis.—W. U., æt, 60. Clerk. Adm. June 1. Disch. June 27. In H. 26 days. *Relieved.* Dr. Garrod, vol. 1, p. 119. Temperate. No family history of gout. First attack, 13 yrs. ago in both great toes. An interval of 2 yrs. between first and second attack. Three or four slighter ones since. Prev. ill. 7 months. General gout commencing in knees. On adm. almost all the joints of extremities stiff and painful. No concretions. No uric acid in serum of blood drawn on the 20th day after adm.

Ammoniated tincture of guiacum and iodide of potassium.

*Gout.—G. G., æt. 48. Cabinet Maker. Adm. Mar. 23 Disch. Ap. 1. In H. 9 days. *Recovery.* Dr, Garrod, vol. 1, p. 78. Moderately temperate. First attack 9 years ago in ball of right great toe. The second 5½ years ago in both feet. 5 years ago a third in both feet. 3 years ago a fourth in hands and elbows. General gout twice in last six months. Prev. ill. 3 days. Right hand, right elbow, left knee, right knee. and feet affected in succession. On adm. right hand and elbow very red and swelled. No concretions. Liver large. Urine albuminous for 3 days after adm. Well on 5th day after adm.

Colchicum wine. Ammoniated tincture of guaiacum.

*Gout.—W. B., æt. 47. Cabman, Adm. Mar. 23. Disch. May 9. In H. 47 days. *Much relieved.* Dr. Beale. Not very temperate. Three attacks of gout in last 7 years, each commencing in right great toe. The last attack 2 yrs. ago. Prev. ill. 9 days. Commenced in ball of right great toe, then in right ankle, right knee, left ankle. No concretion in ears, ball of right great toe enlarged. Convalescent on 32nd day, with exception of slight redness and tenderness about left ankle.

Bicarbonate of potass, opium, sulphate of magnesia, and bark (36 *days*). *Sesquichloride of iron* (5 *days*). *Then bicarbonate of potass.*

Gout.—Disease of Bladder.—A. A., æt. 51, Brass Founder. Adm. Nov. 17. Disch. Nov. 30. In H. 13 days. *Relieved.*—Dr. Beale, vol. 7, p. 9. First attack of gout three years ago. Dysuria and irritable bladder, since acute renal dropsy 2½ years ago. Occasional hæmaturia 12 months. Lumbar pain 2 months. Urine albuminous, containing blood, phosphates, and bladder epithelium. Gout in left foot on 3rd day after adm.

Sesquichloride of iron. Bark, colchicum, bicarbonate of potass, and iodide of potassium. Loins dry cupped.

Gout.—G. P., æt. 44, Paperhanger. Adm. Dec. 17. Disch. Dec. 26. In H. 9 days. *Recovery.*—Dr. Garrod, vol. 3. p. 170. Formerly a House-painter. First attack 16 years ago. Since then lead colic twice, gout frequently. Now ill 2 days. Left thumb painful, anchylosis of ball of left great toe, and partially of left ankle. No concretions. Slight purpura. Well about 10th day.

Guaicum and iodide of potassium.

Gout.—J. P. M., æt. 38, Gunmaker. Adm. Oct. 31. Disch. Nov. 9. In H. 9 days. *Recovery.*—Dr. Garrod, vol. 3, p. 129. Gout three or four times a year for last seven years. Prev. ill. 14 days. In left knee, right foot, right knee, and right thumb. Moderate quantity of uric acid in blood serum. Slight albuminuria. Quite well on 22nd day.

Colchicum, and sulphate and carbonate of magnesia.

Gout.—J. D., æt. 59, Butcher. Adm. Sept. 22. Disch. Oct. 17. In H. 26 days. *Recovery.* Dr. Garrod, vol. 3, p. 35. Not very temperate. Gout frequently during last 20 years. Weakness of left arm, and wasting of muscles from disuse, consequent on gout in wrist, elbow, and shoulder during last 2 months.

Iron. Galvanism. J. P. to hand.

Gout.—W. D., æt. 38, Bootmaker. Adm. Sept. 7. Disch. Oct. 3. In H. 26 days. *Recovery.* Dr. Johnson, vol. 19, p. 46. Intemperate. Father gouty. First attack. Prev. ill. 7 days. In left hand, ankles, right hand, hips, and shoulders. Tongue slightly coated. Skin dry. Well on 26th day.

Bark, bicarbonate of potass, and colchicum. Cotton wool and alkaline lotion to joints.

Gout.—G. D., æt. 42, Coachman. Adm. Sept. 12. Disch. Sept. 28. In H. 16 days. *Recovery.* Dr. Johnson. Acute rheumatism 4 years ago. Pain in chest, shortness of breath, and loss of appetite, 5 days. Right great toe attacked on day before adm., then right foot, ankles, left hand and fingers, right wrist and fingers. Free sweating. Tongue furred. Free from pain on 11th day.

Bark, iodide of potassium, and bicarbonate of potass. Colchicum and opium.

Gout.—G. G., æt. 46, Bootmaker. Adm. Oct. 28. Disch. Dec. 19. In H. 52 days. *Recovery.* Dr. Garrod, vol. 3, p. 115. Mother had gout. Four months ago gout in 2nd and 3rd toes of left foot, right foot, right side, and right arm. Ill 9 weeks. Bladder irritable since. Prev. ill. 7 days. Pain in heels and ankles, back, right elbow and hand, left elbow. Tongue white. Trace of uric acid in blood serum. Free from pain on 35th day—hands afterwards painful.

Iodide of potassium and guaiacum. Quinine Colchicum. Cod-liver oil. E. L. to elbow. J. P. to hands.

Gout.—J. C., æt. 63, Salesman. Adm. July 11. Disch. Aug. 5. In H. 35 days. *Relieved.* Dr. Beale, vol. 5, p. 230. Father gouty. Frequent attacks for 25 years. Prev. ill. 5 weeks. Much effusion into left knee, left ankle, and right little finger also effected. Tongue furred. Concretion in right ear.

Bicarbonate of potass and sulphate of magnesia. Liq. cinchonæ. Blisters to knee.

Acute Rheumatism.—Kate L. æt. 34. Adm. Aug. 7. Disch. Sept. 9. In H. 33 days. *Recovery.* Dr. Garrod, vol. 1, p. 199. First attack 8 years ago. Prev. ill. 1 day. In right ankle, left ankle, knees, and hips. On 4th day right hand and wrist, and right elbow and shoulder attacked. On 6th day in left shoulder. Pain worse at night. Free sweating. Tongue coated. Violent noisy delirium on 6th evening. Free from pain on 8th day. The delirium began to subside about 19th day, and had quite vanished on 29th day. Heart normal throughout.

Quinine and bicarbonate of potass. Opiates and stimulants. Nitric acid and bark during convalescence.

*Acute Rheumatism.—A. R., æt. 14. Adm. Nov. 7. *Died* Nov. 30. Dr. Garrod, vol. 4, p. 2. First attack a year ago. Prev. ill. 3 days. Began with palpitation and cardiac pain. Then left knee and ankle, right knee and ankle, and arm joints, attacked in turn. On adm. loud systolic bruit over apex, and constant severe præcordial pain. Tongue furred. Joints well on 11th day. On 18th day crep. and dulness at base of right lung, and slight albuminuria (6 days). On 19th day fresh pain in shoulders. On 21st day dulness and crep. at both bases. On 23rd day dulness, bronchial breathing, and bronchophony over both sides behind, nearly to scapular spine on right side. On 25th day P. 140, R. 80. On 27th day P. irregular, 136. Death from syncope. P. M.—Lower pulmonary

lobes nearly hepatized. Pericardium universally adherent. Beads of lymph on aortic valves. Left vent. hypertrophied.

Quinine and bicarbonate of potass. Bark and carbonate of ammonia. Brandy up to 12 *oz. Subdermic inj. of morphia reg. cord.*

Acute Rheumatism.—A. R., æt. 22, Compositor. Adm. Sept. 11. Disch. Oct. 17. In H. 36 days. *Recovery.* Dr. Garrod, vol. 3, p. 8. Four or five attacks since the first, 15 years ago. Prev. ill. 8 days. In back of neck, left arm and shoulder consecutively. Tongue slightly furred. Free from rheum on 16th day, with exception of slight pain in neck. On 22nd day slight epistaxis and hæmoptysis, and much blood in motions for a few days. Has phthisis.

Bicarbonate of potass and quinine (6 *days*). *Turpentine and gallic acid for hæmorrhage. Quinine and cod-liver oil.*

Acute Rheumatism.—Eliza N., æt. 17, Servant. Adm. Sept. 14. *Died.* Sept. 21. In H. 7 days. Dr. Johnson. First attack. Prev. ill. 2 days. In back of neck, left hip, shoulder, elbow, wrist, ankle, and knee, then in same joints of right side. Profuse sweating. Yellowish brown fur on tongue. Systolic bruit over heart. On 7th day rhonchus and sibilus over lungs, and crepitation at bases. Albuminuria. Death on 10th day. P.M.—Recent thickening of mitral valve. Bronchial mucous membrane inflamed. Congestion of lower lobes of lungs.

Acute Rheumatism.—W. G. æt. 31. Adm. July 28. *Died* on July 29. Dr. Johnson. Had been ill a week. On adm. delirious. Extremities cold. P. thready, 146. Profuse sweating. Pain in hips. To and fro sound over heart.

Acetate of potass, carbonate of ammonia, chloric ether, and quinine and opium. Brandy 24 *oz.*

*Acute Rheumatism.—H. B. æt. 72, Surgical Instrument Maker. Adm. Nov. 25. *Died* on Dec. 12. In H. 17 days. Dr. Johnson, vol. 10, p. 167. First attack. Prev. ill. about 14 days. First left ankle, then right ankle, red swelled, pitting and painful. Purpuric spots on legs. On 29th day ankles free from pain, but very œdematous, and albumen, blood, renal epithelium, and waxy and granular casts in urine. On 31st day extreme tenderness over right kidney. P. 36. Tongue brown. Sibilus over lungs. Death on 32nd day. P.M.—Fluid in thoracic cavities. Lungs œdematous. Aorta atheromatous and dilated. Right kidney large, congested, studded with hæmorrhagic spots and small cysts.

Bicarbonate of potass and iodide of potassium (12 *days*). *Quinine and sulphuric acid* (3 *days*). *Aromatic spirits of ammonia and decoction of bark* (2 *days*). *Sinapism to loins. Purgatives.*

*Acute Rheumatism.—W. B., æt 32, Timekeeper. Adm. Oct. 20. *Died.* on Dec. 11. In H. 52 days. Dr. Garrod, vol. 3, p. 98. Eighth attack. The first, 14 years ago. Prev. ill. 6 days. Left knee, left ankle, right knee, right ankle, right wrist and knuckles, left wrist and knuckles, shoulders, and tempors maxillary joints affected in turn. Tongue white. Systolic bruit over apex on adm. On 11th day joints well, crepitation below left nipple and angle of scapula, dulness and crepitn. at right base. Sputa rusty on 18th day. Albuminuria on 19th day. Diarrhœa. On 46th day dulness below right scapula, behind, coarse crepitation all over lungs; sputa still bloody. On 58th day *delirous*, p. 160. Death on 59th day. P.M.—Patch of pulmonary apoplexy in right lower lobe. Pericardium somewhat adherent. Mitral orifice contracted. Beads of lymph on thickened edge of valve. Albuminous (?) degeneration of lower half of left kidney.

Bicarbonate of potash and quinine (2 *days*). *Acetate and carbonate of ammonia* (17 *days*). *Carbonate of ammonia* (18 *days*). *Bark and ammonia. Brandy to* 18 *oz. Astringents and opium for diarrhœa.*

*Acute Rheumatism.—W. B., æt. 27, Carpenter. Adm. June 3. Disch. July 22. In H. 49 days. *Recovery.* Dr. Beale, vol. 5, p. 178. First attack a

year ago. Prev. ill. 5 days. Left wrist, left shoulder, ankles and knees affected consecutively. Joints free from pain on 26th day. Return in shoulders. Finally well on 36th day. Heart normal on adm. Pericardial friction sound on 9th day (8 days). Cardiac dulness increased till 13th day, normal on 17th day. Systolic bruit after 22nd day. On 9th day dulness and small crepitn. at *right* base, afterwards œgophony, pleuritic rub, and absence of vocal vibration. On 15th day bronchial breathing (13 days). On 12th day dulness and crepitn. below *left* scapular angle. Br. Br. (17 days) on 15th day extending up to spine on 19th day. Lungs clear on 41st day. Expectoration untinged throughout. Albuminuria on 9th day (5 days).

Bi arbonate of potass (5 days). Liq. ammon. Acetatis (11 days). Sp. ammon. arom. (10 days). Muriatic acid and pepaine. Chloric ether and brandy. Cupped to 4 oz. over rt. side on 11th day. Dry cupping. Turpentine stupes.

Acute Rheumatism.—F. P., æt. 19, Adm. Nov. 3. In medical ward 29 days. *Reli ved.* Dr. Johnson, vol. 19, p. 135. Gonorrhœa 3 weeks. Prev. ill. 4 days. On adm. much effusion into left knee, right elbow painful. Tongue furred. Free sweating. Basic pericardial brush. Elbow well on 11th day. About 22nd day knee much larger, erysipelatous patch on inner side, much constitutional disturbance. Transferred to surgeons about Dec. 2.

Bicarbonate and nitrate of potass, and quinine, and opium (8 days). Then same with potass. Iodid. Leeches to knee. Purgatives. Straight splint.

Acute Rheumatism.—E. V., æt. 51, Charwoman. Adm. Sept. 26. Disch. Nov. 14. In H. 49 days. *Recovery.* Dr. Johnson, vol. 19, p. 91. First attack. Father gouty. Prev. ill. 14 days. In right hand knuckles and wrist, left knuckles and wrist, shoulders, right knee and ankle, left ankle. Free sweating. Tongue furred. Dyspnœa for 4 days before adm.; then cardiac pain and tenderness, and to and fro sound at apex. On 19th day only occasional friction sound at base; systolic bruit loudest at apex. Convalescent about 54th day.

Bicarbonate and nitrate of potass, and quinine, and opium. Brandy up to 8 oz. Iron.

Acute Rheumatism.—H. S., æt. 32, Compositor. Adm. Oct. 14. Disch. Nov. 14. In H. 31 days. *Recovery.* Dr. Johnson, vol. 17, p. 126. First attack. Prev. ill. 14 days. In left knee, right knee, ankles, and hips, right shoulder, wrist, and hand successively. Profuse sweating. Tongue moderately furred. First sound of heart prolonged. Free from pain on 42nd day.

Bicarbonate and nitrate of potass, and quinine opium. Iron. Brandy.

Acute Rheumatism.—A. P. 16, Compositor. Adm. July 20. Disch. Sept. 4. In H. 46 days. *Recovery.* Dr. Beale, vol. 6, p. 234. Acute renal epilepsy 9½ months ago. First attack. Prev. ill. 14 days. In left instep, left knee, right ankle, right elbow, left elbow, right knee, hands, wrists, and right sterno clavicular joint. Free sweating. Creamy fur on tongue. Slight albuminuria on adm. Cystine in urine about 37th day. Slight pain in joints on 46th day. Heart unaffected.

Bicarbonate of potass, and liq. ammon. acetatis.

Acute Rheumatism.—W.W., æt. 34, Stone Sawyer. Adm. Oct. 19. Disch. Nov. 11. In H. 23 days. *Recovery.* Dr. Johnson, vol. 17, p. 134. First attack. Prev. ill. 5 weeks. All joints affected. Free sweating. Soft systolic bruit at apex, and faint pericardial brush at base. Free from pain on day before adm. Had been under treatment a fortnight.

Bicarbonate and nitrate of potass, carbonate of ammonia, and quinine and opium.

Acute Rheumatism.—A. H., æt. 28, Glazier. Adm. Aug. 5. Died on Aug. 7. In H. 2 days. Dr. Beale, vol. 5, p. 194. First attack. Prev. ill. 14 days. Began in knees. Most of the joints attacked. On adm. friction sound at base of heart. Delirium. Diarrhœa. Skin hot and dry. Died on 7th.

Bicarbonate and nitrate of potass, with liq. opii. sed., and quinine and opium.

Acute Rheumatism.—M. M'C., æt. 48, Shoemaker. Adm. June 1. Disch. July 15. In H. 44 days. *Recovery.* Dr. Johnson, vol. 16, p. 229. First attack. Prev. ill. 12 days. In right elbow, right hand, left hand and shoulder, toes and and ankles successively. Tongue thickly coated. Profuse sweating. Pericardial friction sound on 15th day. Free from pain on 22nd day. Slight return in shoulders and feet. Finally free from pain on 45th day.

Bicarbonate of potass and carbonate of ammonia, with quinine and opium. Brandy to 8 *oz. Opiates. During relapse acetate of potass instead of bicarbonate.*

Acute Rheumatism.—R. T., æt. 39, Keeper at an asylum. Adm. June 8. Disch. July 22. In H. 44 days. *Recovery.* Dr. Johnson, vol 17, p. 47. Fourth attack. The first 16 years ago. Gout 13 months ago. Prev. ill. 5 weeks. Ankles, knees, shoulders, elbows, and wrists attacked in succession. Profuse sweating. Brown fur on tongue. Friction sound over heart's base from 38th to 52nd day. Much crepitus at bases of lungs about 47th day. Joints well on 45th day. Relapse (10 days) about 66th day.

Bicarbonate of potass and quinine and opium (16 *days*). *Brandy to* 8 *oz.*

Acute Rheumatism.—A. C., æt. 26, Porter. Adm. Nov. 24. Disch. Dec. 15. In H. 21 days. *Recovery.* Dr. Garrod, vol 3, p. 160. Gout 8 years ago. Ankles swelled 1 mo., painful 14 days. In left ankle, right ankle, knee and hip successively. Free sweating. Tongue coated. Systolic bruit at apex on adm. Crepitus at bases of lungs. Free from pain on 24th day.

Quinine and bicarbonate of potass. Brandy. Turpentine stupes to chest.

Acute Rheumatism.—P. R., æt. 25, Cook. Adm. Nov. 6. Disch. Nov. 21. In H. 15 days. *Recovery.* Dr, Johnson, vol. 17, p. 145. First attack. Prev. ill. 2 days. In right wrist, then in ankles. Free sweating. Sore throat and dysphagia. Free from pain on 8th day.

Chlorate of potass, sesquichloride of iron, and glycerine. Iron and glycerine to throat.

Acute Rheumatism.—R. L., æt. 38, Waiter. Adm. Oct. 21. Disch. Nov. 4. In H. 14 days. *Recovery.* Dr. Johnson, vol. 18, p. 61. First attack 8 yrs. ago. Prev. ill. 14 days. Ankles and right knee affected. Tongue white. Free from pain on 24th day. Headless tapeworm expelled after a dose of malefern oil.

Bicarbonate of potass, with iodide of potassium and bark. E. L. to knee.

Acute Rheumatism.—Alice S., 10. Adm. Oct. 31. Disch. Nov. 18. In H. 18 days. *Recovery.* Dr. Johnson, vol. 17, p. 77. Second attack. Prev. ill. 5 days. Ankles, knees and left wrist affected successively. Basic systolic bruit. P. 92. Free from pain on 18th day of illness.

Bicarbonate and nitrate of potass, and quinine and opium.

Acute Rheumatism.—W. P., 18, Shopman. Adm. Oct. 20. Disch. Oct. 31. In H. 11 days. *Recovery.* Dr. Johnson, vol. 18, p. 51. First attack a year ago, slight. Prev. ill. 3 wks. All joints attacked before adm. Then knees painful. Tongue furred. Mitral systolic bruit at apex. Free from pain about 22nd day.

Bicarbonate of potass, quinine, and opium.

Acute Rheumatism.—Eliza B. 21, Servant. Adm. Oct. 28. Disch. Nov. 18. In H. 21 days. *Recovery.* Dr. Beale, vol. 5, p. 334. First attack. Prev. ill. 14 days. In back, knees, ankles, right wrist, left wrist, elbows, shoulders, successively. Free sweating. Tongue slightly furred. Soft systolic bruit at apex, and pericardial friction sound on adm. Free from pain on 23rd day.

Bicarbonate of potass and sp. ammon. arom. (13 *days*). *Brandy* 4 *oz.*

Acute Rheumatism.—Mary R., æt. 30, Cook. Adm. June 10. Disch. July 18. In H. 38 days. *Recovery.* Dr. Garrod, vol. 1, p. 136. First attack 9 years ago,

heart affected. Second 3 months back, more or less since. Prev. ill. 5 days. Hands, elbows, shoulders, ankles, knees affected successively. Tongue coated. Much sweating. Old mitral systolic bruit. On 9th day sputa slightly bloody, and dulness crepitus, and a little bronchial br. at bases. Then free from pain, but slight return afterwards in left wrist.

Quinine and bicarbonate of potass. Opiates nightly. Hirud. viij. reg. cord.

Acute Rheumatism.—Fanny H., æt. 22, Servant. Adm. Oct. 14. Disch. Nov. 11. In H. 28 days. *Recovery.* Dr. Garrod, vol. 3, p. 77. First attack 10 years ago. Prev. ill. 6 days. In right knee, right hand, elbow and shoulder, and right ankle, then in joints of left side in same order successively. Free sweating. Tongue slightly furred. Systolic bruit over apex on adm. Friction sound over base on 8th day. Free from pain on 28th day.

Bicarbonate of potass and quinine (22 days).

Acute Rheumatism.—J. D., æt. 17, Housemaid. Adm. Oct. 20. Disch. Nov. 14. In H. 25 days *Recovery.* Dr. Garrod, vol. 3, p. 104. First attack. Prev. ill. 7 days. In ankles, knees, elbows, and wrists successively. Basic pericardial friction sound on adm. Tongue furred. Skin hot. Much pain in chest (till 22nd day). Joints well on 15th day.

Quinine and bicarbonate and acetate of potass (16 days). Brandy.

Acute Rheumatism.—E. C., æt. 29, Ostler. Adm. June 13. Disch. July 8. In H. 25 days. *Recovery.* Dr. Johnson, vol. 15, p. 247. First attack 8 yrs. ago. Lead colic 4 yrs. ago. Prev. ill. 4 days. In right ankle, left ankle, knees, left shoulder, and wrists successively. Profuse sweating. Yellowish white fur on tongue. Bowels confd. Systolic bruit over apex of heart on adm. Free from pain on 20th day.

Acetate of potass and carbonate of ammonia, and quinine and opium. Brandy.

Acute Rheumatism.—G. D., æt. 20, Porter. Adm. Sept. 21. Disch. Oct. 31. In H. 40 days. *Recovery.* Dr. Garrod, vol. 3, p. 31. First attack. Prev. ill. 10 days. In ankles, shoulders, elbows, wrists, fingers, and sterno clavicular joints successively. Tongue furred. Moderate sweating. Systolic bruit over apex on adm. Free from pain on 20th day, but fresh attack on 25th day in ankles, knees, and wrists.

Quinine and bicarbonate of potass.

Acute Rheumatism.—Ann R., æt. 26, Married. Adm. Sept. 30. Disch. Oct. 19. In H. 19 days. *Recovery.* Dr. Garrod, vol 3, p. 46. First attack a year and a half ago. Prev. ill. about 25 days. Left knee and ankle, right knee and ankle, left shoulder, elbow and wrist, and right shoulder, elbow and wrist affected successively. Systolic bruit over heart on adm.; loudest at apex. Tongue furred. Frequent metastasis. Friction sound at base about 25th day. Free from pain on 32nd day.

Quinine and bicarbonate of potass. Brandy.

Acute Rheumatism.—F. A., æt. 16, Housemaid. Adm. Aug. 6. Disch. Sept. 23. In H. 48 days. *Recovery.* Dr. Beale, vol. 5, p. 240. Third attack. First 5½ yrs. ago. Prev. ill. 12 days. In ankles, knees, shoulders, elbows, and wrists successively. Constant præcordial pain. Free sweating. Tongue thickly furred. P. 120. Systolic bruit over apex on adm. Free from pain on 48th day.

Quinine, bicarbonate, and nitrate of potass, and acetate of ammonia.

*Acute Rheumatism.—Alice J., æt. 16, Servant. Adm. June 27. *Died* on Aug. 2. In H. 36 days. Dr. Beale, vol. 5, p. 210. First attack four years ago. Prev. ill. 3 days. Knees, ankles, and right hand painful in turn. Profuse sweating. Thick white fur on tongue. Acute præcordial pain. Slight albuminuria. On 7th day diarrhœa and tympanites (3 days), and crepitation and pleuritic rub at right base. Afterwards dullness and bronchial br. over bases till 32nd day, when dulness, no br. br. or resp. murmur, slight crepitation. Joints well on 12th day.

About 18th day hœmaturia and ædenia of feet and ankles. To and fro sound over heart on adm. About 13th day extension of cardiac dulness to 2nd rib and right sternal edge; disappearance of friction sound. Rub again heard on 14th day, all over heart on 16th day. Great increase of cardiac dulness afterwards. P. M. Recent pleuritic adhesions. Three pints fluid in pericardium. Fresh adhesions about base of heart. Fibrinous warts on mitral valve. Right lung and lower lobe of left dark red and solid.

Bicarbonate of potass, acetate of ammonia and sp. ammon. arom. Afterwards sulphutic ether, sesquichloride of iron, and liquor cinchonæ. Brandy to 16 *oz. Mercurial ointment with opium over heart. Loins dry cupped.*

Acute Rheumatism.—Sarah C., æt. 24, Married. Adm. July 10. Disch. July 24. In H. 14 days. *Recovery.* Dr. Garrod, vol. 1, p. 167. First attack six years ago. Prev. ill. 4 days. Knees, ankles, and hips affected. Moderate sweating. Tongue blankety. P. 104. Free from pain on 11th day.

Quinine and bicarbonate of potass.

Acute Rheumatism.—Eliza K., æt. 25, Servant. Adm. June 1. Disch. July 1. In H. 30 days. *Recovery.* Dr. Johnson, vol. 16, p. 233. First attack 13 years ago. Ankles painful 2 months. Prev. ill. 1 week. Ankles, knees, wrists, shoulders, and elbows successively affected. Profuse sweating. Furred tongue. Slight basic systolic bruit. Free from pain on 19th day.

Bicarbonate of potass and carbonate of ammonia, and quinine and opium.

Acute Rheumatism.—Jane T., æt. 22, Servant. Adm. June 17. Disch. July 1. In H. 14 days. *Recovery.* Dr. Garrod, vol. 2, p. 178. First attack. Prev. ill. 5 days. Ankles, knees, wrists, and elbows attacked. Tongue furred. Systolic bruit over apex on adm. Free from pain on 10th day.

Quinine and bicarbonate of potass.

Acute Rheumatism.—L. K., æt, 16, Kitchenmaid. Adm. Sept. 15. Disch. Oct. 5. In H. 20 days. *Recovery.* Dr. Garrod, vol. 3, p. 16. First attack. Prev. ill. 8 days. Left ankle, knee, elbow, and hand, right shoulder, elbow, and hand successively affected. Free sweating. Tongue furred. First sound of heart prolonged, and slight præcordial pain. Free from pain except in left hand on 23rd day.

Quinine and bicarbonate of potass. Opiates at night. Brandy.

Acute Rheumatism.—D. O., æt. 19, Waiter. Adm. Oct. 30. Disch. Nov. 21. In H. 22 days. *Recovery.* Dr. Garrod, vol. 3, p. 123. First attack 4 years ago. Prev. ill. 4 days. In right ankle and left wrist. Slight sweating. Tongue furred. Free from pain on 9th day. Roughness with second sound about 11th day. P. intermittent on discharge.

Quinine and bicarbonate of potass.

Acute Rheumatism.—T. C., æt. 38, Cabman. Adm. May 18. Disch. July 13. In H. 57 days. *Recovery.* Dr. Johnson, vol. 13, p. 39. First attack 11 years ago. Prev. ill. 1 day. Left knee and ankle, right hand and wrist, and right knee and ankle affected in succession. Profuse sweats. Tongue slightly furred. On 4th day friction sound over heart (20 days), and afterwards a faint systolic bruit. Free from pain on 33rd day. Relapsed on 44th day. Finally convalescent on 53rd day

Bicarbonate of potass and quinine and opium. Cotton wool to joints.

Acute Rheumatism.—Hope C., æt. 28, Married. Adm. Oct. 19. Disch. Nov. 14. In H. 26 days. *Recovery.* Dr. Beale, vol. 5, p. 320. Fifth attack; first 24 years ago. Prev. ill. 3 months. In left great toe, knees, hands, and elbows successively. Return of pain shortly after adm. Tongue furred. Free sweating. Double bruit at base; heart's impulse heaving.

Liquor cinchonæ and bicarbonate of potass. Whisky 6 *oz.*

Acute Rheumatism.—E W., æt. 38, Needlewoman. Adm. Oct. 5. Disch. Nov. 21. In H. 47 days. *Recovery.* Dr. Garrod, vol. 3, p. 59. First attack. Prev. ill. 14 days. On adm. very weak and emaciated, bedsore over sacrum.

P. 124, R. 36. Cardiac dulness increased in area; to and fro sound at apex; slight albuminuria. On 3rd day after adm. right shoulder and wrist, left ankle, elbows and knees painful. Joints soon well, but heart's action irregular till 27th day.

Bicarbonate of potass, carbonate of ammonia, and bark. Brandy.

Acute Rheumatism.—A. L., æt. 33, Laundress. Adm. Nov. 17. Disch. Dec. 30. In H. 43 days. *Recovery.* Dr. Johnson, vol. 18, p. 98. First attack. Rheumatic iritis 2 years ago. Prev. ill. 5 weeks. Ankles, wrists, knuckles, and right knee successively affected. Selerotitis about 48th day. On 64th day free from pain except in right ankle. Finally free from pain 2 days before discharge.

Bicarbonate of potass, iodide of potassium, and bark. Opium. E. L. post. aur. dext.

Acute Rheumatism.—S. Ann de K., æt. 15. Adm Aug. 12. Disch. Oct. 16. In H. 65 days. *Recovery.* Dr. Johnson, vol. 17, p. 115. Prev. ill. 4 days. Left foot and knee, right ankle and knee, shoulders, elbows, and wrists successively affected. Thick blankety fur on tongue. Free sweating. Loud basic diastolic bruit. Pericardial friction sound on 7th day, quite disappearing as cardiac dulness increased, again audible on 34th day. About 13th day delirium at night, and dulness and pleuritic rub at left base. Remained very anæmic.

Quinine and bicarbonate of potass (13 *days*). *Then liq. ammon. acetatis and sp. ammon. arom. Iron. Brandy up to* 12 *oz.*

Acute Rheumatism.—Louisa C., æt. 25, Married. Adm. Nov. 14. Disch. Dec. 9. In H. 25 days. *Recovery.* Dr. Beale, vol. 7, p. 1. Had scarlet fever 13 years ago. Three weeks ago sore throat, enlarged ceroical glands, and fever without eruption. Right thumb and arm, back of neck, left arm, knees and ankles affected successively during last 14 days. Free sweating on adm. General cuticular desquamation. Elbows, right shoulder, and knees painful. Tongue slightly furred. Basic systolic bruit. Free from pain on 25th day.

Bicarbonate and nitrate of potass. Occasional nightly opiates.

*Acute Rheumatism—Pyæmia—Erysipelas.—P. K., æt. 21, Policeman. Adm. April 14. *Died* on July 25. In H. 102 days. Dr. Beale, vol. 6, p. 28. Prev. ill. 1 month. Back of neck, legs, and shoulders painful in turn. Free sweating. Tongue furred. About beginning of May abscesses formed in calves of legs, followed by extensive sinuses. Much effusion into left knee about beginning of July, and about July 14 abscesses about right wrist and hand and head of right fibula. On July 17 erysipelas of face, followed by sloughing of skin of forehead and eyelids. Occasional delirium. Dry brown tongue. Fresh abscesses about knees and elbows and palm of left hand. Death.

Alkalies and quinine. Sesquichloride of iron and quinine. Cod liver oil. Pepsine. On July 4. Potass, bicarb., eth. chlor., and sp. ammon. arom. (10 *days*). *Then sp. amm. arom. and chloric ether. Brandy. Abscesses opened.*

Chronic Rheumatism.—A. R., æt. 19½, Warehouseman. Adm. Sept. 22. Disch. Nov. 11. In H. 50 days. *Much relieved.* Dr. Garrod, vol. 3, p. 37. Sequel of acute attack 9 months ago. Now fingers and ankles painful, impaired motion of elbows and right shoulder. On disch. ankles better.

Bark and iodide of potassium. Quinine and iron. Guaiacum. Gallic acid. Cod liver oil. Blisters to ankles.

Chronic Rheumatism.—W. J., æt. 42, Carpenter. Adm. Dec. 9. Disch. Dec. 19. In H. 10 days. *Relieved.* Dr. Beale, vol. 7, p. 27. Five attacks of acute rheumatism, the first 26 years ago. Prev. ill. 2 months. All joints affected. Increased cardiac impulse, and slight diastolic bruit.

Liquor cinchonæ and bicarbonate of potass.

Chronic Rheumatism.—W. R., 49, Coachman. Adm. Nov. 25. Disch. Dec. 16. In H. 21 days. *Very much relieved.* Dr. Johnson, vol. 17, p. 167. Frequent

acute rheumatism since first attack 44 years ago. Flying pains in joints since recovery from last attack 7 weeks ago. Prev. ill. 2 days. Left index, knuckle, and right hip painful and swollen. Slight sweating.

Bicarbonate of potass and iodide of potassium. Iron and colchicum afterwards added.

Chronic Rheumatism.—G. P., æt. 33, Warehouseman. Adm. Nov. 18. Disch. Dec. 28. In H. 40 days. *Improved.* Dr. Beale, vol. 6, p. 343. Acute rheumatism 4 years ago. Afterwards pain in hips. In various hospitals since. Now thighs painful and cannot be quite straightened on trunk. Has albuminuria.

Bark and iodide of potassium. Alkaline baths.

Subacute Rheumatism.—H. P., æt. 26, Cook. Adm. July 17. Disch. Aug. 26. In H. 40 days. *Recovery.* Dr. Garrod, vol. 1, p. 181. Ill three weeks. In elbows and left instep. On adm. pain and swelling of right elbow, extending up and down arm. Moderate sweating. Tongue furred. Basic systolic bruit. Free from pain on 8th day after adm. Elbow stiff for some time.

Guaiacum and iodide of potassium. E L. to elbow. Passive motion.

Subacute Rheumatism.—Mary A. R., æt. 24, married. Adm. July 9. Disch. July 22. In H. 13 days. *Recovery.* Dr. Garrod, vol. 1, p. 169. Third attack of rheumatism. First 8 years ago. Left ankle painful 5 months, stiff and swollen 2 months. Right hand painful 1 week. Left ankle and lower half of leg œdematous. On disch. scarcely any pain, ankle useful.

Quinine and bicarbonate of potass.

Subacute Rheumatism.—E. O., æt. 21, Housemaid. Adm. Sept. 24. Disch. Oct. 24. In H. 30 days. *Recovery.* Dr. Beale, vol. 5, p. 294. First attack. Prev. ill. 1 week. Right knee, left knee, left wrist and hand, right elbow, right ankle, and right knee (2nd time) attacked in turn. Sweating scanty. Tongue slightly furred, free from pain after 20th day.

Bicarbonate and nitrate of potass, and quinine and opium.

Rheumatoid Arthritis—H. A., æt. 53, Widow. Adm. Oct. 13. Disch. Oct. 24. In H. 11 days. *Relieved.* Dr. Garrod, vol. 3, p. 73. Ill 6 years. In right knee, hand, and shoulder. About 1½ years ago joints of left side affected in same order.

Guiacum, bark, and iodide of potassium.

Rheumatoid Arthritis.—Esther L., æt. 23, married. Adm. Oct. 29. Disch. Nov. 11. In H. 14 days. *Much relieved.* Dr. Garrod, vol. 3, p. 119. Prev. ill. 6 weeks. Wrists, finger-joints, right shoulder, elbows, and ankles stiff, painful, and worse at night. Has phthisis; right apex affected. Ankles improved.

Liq. cinchonæ, and iodide of potassium; then guaiacum and bark. Cod liver oil.

Rheumatoid Arthritis.—C. D. æt. 36, Monthly Nurse. Adm. June 26. Disch. Aug. 1. In H. 36 days. *Very much relieved.* Dr. Beale, vol. 6, p. 212. Prev. ill. 9 months. Laid up most of that time. Worse last fortnight. First in right great toe and ankles. On adm. right shoulder, elbow, hand, and wrist; left elbow, hand, knee, and ankle affected. Sweating freely. Creamy fur on tongue. Nearly free from pain on 19th day after adm.

Sp. ammon. arom. and Liq. ammon. acetatis. Opiates at night. Aperients.

Inflamed Knee.—Rhoda V., æt. 37, Servant. Adm. July 31. Disch. Aug. 12. In H. 12 days. *Recovery.* Dr. Garrod. Inflammation of right knee joint 3 weeks. Free from pain on Aug. 11.

Iodine paint. Passive motion. Infusion of quassia.

Diseases of Knee.—M. M., æt. 17, Kitchenmaid. Adm. Dec. 28. Disch.

Dec. 31. In H. 3 days. Dr. Johnson. Pain and great swelling of right knee, 1 week. Scrofulous Keratitis 3 weeks. Transferred to surgeons Dec. 31.

Gonorrhœal Rheumatism.—J. J. æt. 22, Clerk. Adm. Nov. 9. Disch. Dec. 12. In H. 33 days. *Recovery.* Dr. Garrod, vol. 1, p. 228. Gonorrhœa well 8 or 9 days ago. Prev. ill. 10 days. In left knee, hip, and sole of foot. Tongue slightly furred. First sound of heart prolonged. Hip and heel still slightly painful on 40th day.

Guaiacum and iodide of potassium. Asafœtida. Subdermic injection of morphia. Hot air baths.

Sciatica.—R. B., 40, Brickmaker. Adm. Sept. 1. Disch. Sept. 23. In H. 22 days. *Recovery.* Dr. Johnson. Second attack. First slighter, 12 months ago. Prev. ill. 10 weeks. Sudden severe pain from left loin to ankle. Loin tender. Leg numb. Free from pain on 20th day after adm.

Lumbago.—A. P., æt, 27, Footman. Adm. June 20. Disch. July 11. In H. 21 days. *Nearly well.* Dr. Johnson. Pain in loins very severe at night, and constipation 2 months. Oxaluria.

Aperients. Quinine and iron. Iodide of potassium and iron. Opiates. Hot air baths.

Pericarditis.—J. H., æt. 54, Labourer. Adm. July 21. Disch. Aug. 19. In H. 29 days. *Recovery.* Dr. Johnson, vol. 19, p. 26. Never had rheumatism. Pain in left shoulder 14 days. Pain in left side, dyspucea, and nocturnal palpitation, 2 days. On adm. cardiac dulness much enlarged, and loud to and fro sound over heart. P. 100, R. 32. Dulness and crepitation under left clavicle, and at left posterior base. 10 days later heart's sounds and dulness normal.

Carbonate of ammonia and liquor ammon. acetatis (7 days). Then quinine and sesquichloride of iron. Brandy, 4 *oz.*

*Pericarditis.—T. K., æt. 46, Porter. Adm. Dec. 10. Disch. Dec. 30. In H. 20 days. *Recovery.* Dr. Johnson, vol. 19, p. 182. Intemperate. Typhus 10 years ago. "Caught cold" 2 weeks ago. Prev. ill. 3 days. On adm. pain and tenderness in left hypochondrium, cardiac dulness from 2nd rib to lower border of 6th, and to right of sternum. Friction (to and fro) sound near left nipple, very distinct, 7 days later, inaudible 11 days later. Dulness over lower part of left lung, fine crep. below the 5th rib, in front. Pleuritic rub, harsh br., and increased vocal thrill at angle of scapula. P. 120, R. 30. Expectoration scanty and untinged.

Carbonate of ammonia, chloric ether, quinine and opium (15 *days.*) *Then quinine and sesquichloride of iron. Brandy,* 6 *oz.*

Pericarditis.—H. F., æt. 39, Picture Frame Maker. Adm. Oct. 19. *Died* on Nov. 2. In H. 14 days. Dr. Garrod, vol. 3, p. 93. Cough and dyspnœa, at times, 12 years. Hæmoptysis 16 days ago. On adm. pallid. Systolic bruit over apex of heart; P. 96, R. 32; delirium at night. 3 days later loud to and fro sound over whole cardiac region (till death); 4 days later dulness and br. br. below angle of left scapula; 5 days later tongue dry, much delirium. P.M.—Bloody serum in pericardium. Much recent lymph on heart. Mitral orifice contracted. Left ventricle enormously thickened. Kidneys atrophied (gouty).

Lobelia, henbane and ether (3 *days). Then carbonate of ammonia and citrate of iron and quinine. Turpentine stupes. J. L. to præcordia. Brandy* 6 *oz.*

*Aneurism of Aorta.—J. H., æt. 31, Hawker of playbills. Adm. July 16. *Died* July 20. In H. 4 days. Dr. Johnson, vol. 19, p. 7. Intemperate. Has to run much. Suddenly lost voice 7 weeks ago. Dysphagia 8 days. Cough dyspnœa, and increased hoarseness, 5 days. On adm. voice husky. Breathing

stridulous. No bruit. Laryngoscopic examination showed larynx healthy. Pain at lower end of sternum ; 2 days later slight hœmoptysis ; 2 days later sudden faintness, vomiting of blood twice, and death. P.M.—An aneurism of descending arch, as large as an orange, opening into œsophagus by a valvular aperture the size of a florin. Recurrent laryngeal compressed by it. Stomach full of blood.

Quinine. Gallic acid.

Aneurism of Aorta.—Mary A. O., æt. 32, married. Brought in dead on Sept. 14. Dr. Johnson. Fell down in street. Had been dyspeptic 4 or 5 months. P.M.—A small aneurism of arch of aorta, at junction of ascending and transverse portions, the sac between the aorta and pulmonary artery. Hemorrhage into pericardium through a triangular aperture in left side of aneurism.

Cardiac Disease.—M. A. M., æt. 29, Needlewoman. Adm. Nov. 16. Disch. Nov. 28. *Relieved.* Dr. Johnson, vol. 17, p. 153. Intemperate. Ill 2 years. Palpitation and stabbing pain in præcordial region. Has spat blood. Cardiac dulness increased. Systolic bruit loudest at base. P. 78 feeble. Dulness and wavy expiration at left apex.

Iron and quinine.

Cardiac Disease.—Emma B., æt. 27, married. Adm. Oct. 3. *Died* on Oct. 3. Dr. Garrod, vol. 3, p. 54. Acute rheumatism 6 months ago. Dropsy 3 months. On adm. almost moribund, face blue ; great dyspnœa ; cardiac dulness increased ; double bruit at base ; urine ¼th albumen. P.M.—Fluid in pleuræ pericardium and peritoneum. Heart large ; mitral valve roughened with fibrinous deposits ; warty growths on aortic valves. Lungs œdematous. Kidneys wasted.

Liq. ammon. Acetatis, acetate of potass, and tincture of belladonna.

Cardiac Disease.—Joseph H., æt. 36. Adm. Dec. 9. *Died* Dec. 9. Dr. Johnson, vol. 19, p. 189. Very sallow. Systolic bruit over apex of heart. Died of syncope a few minutes after adm. P.M.—General emphysema of lungs. Heart large ; left ventricle thickened and dilated ; mitral valve incompetent ; aortic orifice and arch much dilated. Kidneys wasted.

Cardiac Disease.—J. W., æt. 32, Porter at Gas Works. Adm. Dec. 8. Disch. Dec. 19. In H. 11 days. Dr. Beale, vol. 8, p. 10. Drinks freely. Never had rheum. Winter cough 5 years. Jaundice 15 months ago. Palpitation and dyspnœa 6 months. Slight œdema of legs ; pain over heart ; cough and dyspnœa. Cardiac dulness increased ; pulsation in scrobiculus ; action irregular. Rhonchus and sibulus over lungs. P. 102. Pain, tenderness, and enlargement of liver ; some yellowness of conjuctivæ ; urine albuminous. Discharged at his own request.

Carbonate of ammonia, chloric ether and senega. Jalap and scammony. Back dry cupped. Brandy 6 *oz.*

Cardiac Disease.—Mary R., æt. 34, Street Hawker. Adm. Oct. 27. Disch Dec. 2. In H. 36 days. *Relieved.* Dr. Johnson, vol. 18, p 69. Never had rheumatism Hæmoptysis 6 months ago. Cough and slight hæmoptysis since. Loss of flesh and strength 3 months. Anæmic Pulse very feeble. Rough systolic bruit and thrill at union of 2nd left rib with cartilage ; soft double bruit at base. Dulness and large crepitation at apex of left lung. Fingers clubbed.

Sesquichloride of iron. Morphia at night.

Cardiac Disease.—W. B., æt 21, Labourer. Adm. July 17. Disch. Aug. 29. In H. 43 days. *Relieved.* Dr. John on, vol. 19, p. 12. Acute rheumatism 8 years ago. Palpitation 2 years. Three weeks ago violent palpitation, pain in chest dyspnœa. and hæmoptysis (½ pt.). Has spat blood since. On adm. Florid. Has orthopnœa. R. 28 easily hurried. Dulness and fine crepitation over base of right lung. Heart's apex between 6th and 7th ribs 1½ in. to left of nipple ; strong impulse here and in scrobiculus. Systolic bruit loudest at apex, and loud diastolic bruit at base. P. full, collapsing, 120. Expectorates much dark blood.

Had a second attack of hæmoptysis on day after adm. Still a trace of blood in expectoration when discharged at own request.

Dilute sulphuric acid, chloric ether, opium, and digitalis. Turpentine. Quinine and sesquichloride of iron. Dry cupping to chest.

CARDIAC DISEASE.—J. B., æt. 35, Warehouseman. Adm. Aug. 5. Removed by relatives Aug. 9. In H. 4 days. *Relieved.* Dr. Garrod, vol. 1, p. 195. Acute rheum. 11 years ago. Cough and loss of strength 5 months. Pain in chest and palpitation 4 months. Œdema of legs and great dyspnœa 3 weeks. Very anœmic. Cardiac region extremely tender. Impulse heaving. Systolic bruit at apex and diastolic bruit at base. P. feeble and irregular, 108. R. 34. Urine very albuminous.

Dilute hydrocyanide acid, sulphuric ether and digitalis.

CARDIAC DISEASE.—J. M. M., æt. 54, Porter. Adm. June 23. Disch. July 15. In H. 22 days. *Relieved.* Dr. Garrod, vol. 1, p. 153. Intemperate. Never had rheumatism. Cough and expectoration 5 months. Loss of appetite 3 months. Shortness of breath and palpitation 2 months. Copious rusty expectoration 3 days. Very anœmic. Legs slightly œdematous. Impulse of heart feeble. Double bruit over base. P 88, collapsing, and locomotive. Slight dulness below spine of right scapula. Crepitation at base. Abundant bright rusty expectoration. Gout in lest great toe 6 days later.

Carbonate of ammonia and ammonic citrate of iron.

CARDIAC DISEASE.—Caroline A., æt 53 Bootbinder. Adm. July 8. Disch. July 24. In H. 17 days. *Relieved.* Dr. Beale. vol. 6, p. 226. Never had rheumatism. Cough 20 years. Shortness of breath and palpitation 2 years. Weakness and emaciation 3 weeks. Heart's action irregular. Apex to left of left nipple. Cardiac dulness enlarged. Pulsation in scrobiculus. Systolic bruit at apex. P. 120, small, and irregular. Sibilus and crepitation over chest. Cough and scanty expectoration. Appetite bad. Sleeps badly.

Digitalis and chloric ether. Sal volatile and chloric ether.

CARDIAC DROPSY.—J. T., æt. 53, Butcher. Adm. June 20. *Died* on July 16. In H. 26 days. Dr. Garrod, vol 1, p. 148. Had acute rheumatism when 7 years old. Winter cough 10 years; worse 3 months. Legs dropsical 1 month; inflamed 5 days. Has marked arcus senilus; conjunctivæ yellow. Heart's sounds distant, action irregular. P. 116. No bruit. Crepitation over bases of lungs. Dulness at right base. Urine free from albumen throughout. Sloughing of inner side of right leg. Dyspnœa increased. Death from exhaustion.

Bark and carbonate of ammonia (7 days). Then carbonate of ammonia, citrate of iron, and d coction of senega. Turpentine stupes.

CARDIAC DROPSY.—R. S., æt. 50, Barrister's clerk. Adm. Nov. 5. *Died* on Dec. 5. In H. 30 days. Dr. Johnson, vol. 19, p. 142. Never had rheumatism. Intemperate. Prev. ill. 5 weeks. On adm. Pallid. Legs, thighs, and scrotum œdematous. Soft systolic bruit at apex of heart. Afterwards much ascites, hæmaturia delirium and death. P.M. Recent lymph at lower and back part of right pleura. Heart small. Tricuspid valve thickened. Kidneys pale.

Iron, quinine, and various diuretics. Hydragoque purgatives.

CARDIAC DROPSY.—Susan B., æt. 40, Married. Adm. Sept. 14. *Died* on Nov. 28. In H. 75 days. Dr. Johnson vol. 19, p. 59. Acute rheumatism 8 years ago. Legs œdematous about 2 months, had been much swelled before confinement 3 months ago. Slight ascites. Loud systolic bruit over heart's apex. P. 80. Dulness and crepitation below scapulæ. Albumen and waxy casts in urine. 60 days later much crepitation over lungs, dropsy much increased, legs punctured (twice). Slight improvement. Gangrene of legs. Death. P.M. Some patches of pulmonary apoplexy. Cragula in pulmonary artery. Mitral valve much thickened. Tricuspid orifice dilated. Kidneys wasted. Liver slightly cirrhosed. Many gall stones.

Quinine, iron, and stimulants. Legs punctured. Loins dry cupped.

*Cardiac Dropsy.—B. S., æt. 29, Street Hawker. Adm. Nov. 18. *Died* Dec. 18. In H. 30 days. Dr. Johnson, vol. 17, p. 155. Shortness of breath, cough, and palpitation 4 months. Œdema of legs 1 week. Never had rheumatism. On adm. cardiac dulness 4 in. transversely. Apex to the left; impulse strong. Systolic bruit at apex loudest to left of ensiform cartilage. Soft diastolic bruit over base. P. 110, small. Cough, dyspnœa, and viscid expectoration. Rhonchus and crepitus over back of lungs, dulness at bases. Liver tender. Urine slightly albuminous. 11 days later severe pain about right nipple; great dyspnœa. 2 days later pleuritic rub below nipple. 1 day later dulness below angle of right scapula. 13 days later dulness below 4th rib in front and spine of scapula on right side; crepitation behind; sputa rusty; urgent dyspnœa. 2 days later much pain about nipple; more dyspnœa; delirium; death. P.M. General anasarca. Right pleura full of fluid; lymph on diaphragmatic and pericardial layers. Right lung carnefied; its lower lobe contained patches of circumscribed pulmonary apoplexy. Heart 27 oz. Aortic arch atheromatous and dilated. Pouch over one semilanar valve. Ventricles and auric-ventricular orifices much dilated. Margin of tricuspid valve thickened.

Ammonia and ether. Brandy. Henbane and Indian hemp. Purgatives. Chest dry cupped.

Cardiac Dropsy.—M. A, B., æt. 45, Nurse. Adm. Oct. 12. *Died* on Oct. 17. In H. 5 days. Dr. Garrod, vol. 3, p. 70. Palpitation and œdema of legs on and off 2 years. Hæmatemesis 18 months ago. On adm., anasarca, slight ascites. Systolic bruit loudest at apex. Crepitus at bases of lungs. liver much enlarged and tender, conjunctiva yellow. P. 120, R. 40. Appetite bad. 3 days later delirious. Died of syncope. P.M. Heart fatty. Liver large and fatty.

Dilute muriatic acid, chloric ether, and bark. Carbonate of ammonia. Brandy.

*Cardiac Dropsy.—W. H., æt. 56, Millwright. Adm. Sept. 19. *Died* on Nov. 1. In H. 43 days. Dr. Johnson, vol. 19, p. 72. Very intemperate. Cholera 28 years ago. Typhus 23 years ago. Cough and hoarseness 2 years. Palpitation 12 months. Dropsy 14 days. On adm. conjunctiva slightly yellow; cough raucous. Physical signs of pulmonary emphysema. Musical bruit at apex, systolic, and prolonged into diastole; rough bruit at base. Dulness over base of right lung; bronchial br. about angle of scapula. Hæmoptysis 4 days later. Dulness over left base 2 days later. Urine slightly albuminous 24 days later. P.M. Lungs gorged and emphysematous. Heart 15 oz. Mitral and tricuspid orifices dilated. Trachea compressed by an almost aneurismal dilatation of aortic arch.

Stimulants. Diuretics. Hydragoque purgatives. Brandy to 15 *oz. Chlorodyne. Iron and bark. Legs punctured twice.*

*Cardiac Dropsy.—A. V., æt. 52, Needlewoman. Adm. June 20. Disch. Sept. 30. In H. 102 days. *Recovery.* Dr. Garrod, vol. 2, p. 190. Had scarlet fever 4 times. Acute rheum. 22 years ago. Subject to bronchitis 10 years. Typhoid fever 1½ years ago. Cough and shortness of breath 3 months. Palpitation 2 months. Dropsy of legs 14 days. On adm. considerable œdema of legs. Slight ascites. Dulness below spine of right scapula and some crepitation; elsewhere sibilus. Heart irregular; soft systolic bruit at apex. P. 80, R. 28. Urine slightly albuminous. 31 days later urine 100 oz. daily; dropsy became much less, and ultimately disappeared.

Carbonate of ammonia and iodide of potassium, and bitartrate of potass and jalap powders. Digitalis. Afterwards iron calumba and digitalis.

Cardiac Dropsy.—R. B., æt. 32, Traveller. Adm. June 29. Disch. July 18. In H. 19 days. *Much relieved.* Dr. Garrod, vol. 2, p. 211. Intemperate. Never had rheum. Typhus 18 years ago. Palpitation, headache, and vomiting occasionally for 12 months. Weakness, œdema of legs, cough, and constipated bowels, 5 weeks. On adm. very pallid, legs slightly œdematous. Loud systolic bruit at apex audible all over chest. Dulness and diminished vocal vibration below spine of right scapula. Urine ¼th albumen.

Carbonate of ammonia and digitalis. Bitartrate of potass and jalap.

Cardiac Dropsy.—E. T., æt. 18, Housemaid. Adm. Nov. 21. Disch. Dec. 12. In H. 21 days. *Recovery.* Dr. Johnson, vol. 18, p. 111. Acute rheumatism 10 years ago. Palpitation and shortness of breath on exertion 6 weeks. Œdema of legs 7 days. Heart's impulse strong. Systolic bruit at base, and a softer one at apex. P. 112. feeble. Is anœmic.

Sesquichloride of iron and quinine. Compound jalap powder.

Endocarditis.—E. S., æt. 39, Hospital Nurse. Adm. Nov. 11. Disch. Nov. 26. In H. 15 days. *Recovery.* Dr. Beale, vol. 5, p. 353. Prev. ill. 1 day. Sudden præcordial pain and symptoms of angina. Feverishness. Rough systolic bruit over base of heart faintly audible 12 days later.

Liquor ammoniæ acetatis, liq. cinchonæ, and sp. ammon. arom. (6 *days*). *Then dilute muriatic acid and decoction of bark. Morphia at night.*

Angina Pectoris.—G. A., æt. 62, Tailor. Adm. Aug. 27. *Died* on Aug. 27. Dr. Garrod, vol. 2, p. 268. Picked up in street by police. On adm. face pale, lips blue, severe pain at heart, pulse imperceptible. Left leg and thigh red, swelled, and painful Heart's sounds feeble; 2nd inaudible at apex; faint basic diastolic bruit. Has drunk hard. Cardiac pain and uneasiness some years. At 7 p.m. had an attack of dyspnœa, and died in a few minutes. P.M.—Lungs gorged. Heart flabby, 11¾ oz. Aortic valves atheromatous and incompetent. Coronary arteries ossified. Liver and kidneys large.

Aromatic spirits of ammonia, ether, and tinct. card. co.

Acute Bronchitis.—W. T., æt. 66, Commissionaire. Adm. Sept. 21. Disch. Oct. 10. In H. 19 days. *Recovery.* Dr. Johnson, vol. 17, p. 93. Cough 3 months. Dyspnœa 1 week. On adm. headache, muscular cramps, cough, and mucous expectoration. Rhonchus and sibilus over chest, Tongue furred. P. 100. Highest resp. 32.

Chloric ether and carbonate of ammonia. Chlorodyne. Turpentine stupes.

Acute Bronchitis.—S. G., æt, 58, Charwoman. Adm. Oct. 8. *Died* Oct. 9. Dr. Garrod. Had rheumatism. Breath short for some years. On adm. very weak, pulse small, lips purple. Bedsore over sacrum. Rhonchus and large crepitation over lungs. P.M.—Aortic valves thickened. Mucous membrane of small bronchial tubes intensely inflamed.

Ammonia and chloric ether. Brandy 8 *oz.*

Acute Bronchitis.—C. W., Hospital Nurse. Adm. Dec. 22. Disch. Dec. 31. In H. 9 days. *Recovery.* Dr. Beale. Prev. ill. 1 day. On 3rd day P. 120, R. 36. Rhonchus and sibilus over back of chest. Yellow puriform expectoration.

Aromatic spirits of ammonia, chloric ether, squills, and bark.

Bronchitis—Dropsy.—Selina S., æt. 50. Adm. Oct. 29. Disch. Nov. 11. In H. 13 days. *Recovery.* Dr. Beale, vol. 5, p. 340. Ill 9 weeks. Cough and frothy mucous expectoration. Dyspnœa; œdema of legs. Dulness and crepitation at posterior bases; rhonchus and sibilus elsewhere. P. 80, R. 48. Area of cardiac dulness increased, and first sound prolonged.

Carbonate of ammonia, chloric ether, and senega. Scammony and jalap powder. Turpentine stupes.

Bronchitis.—A. C., æt. 35, Tailoress. Adm. Oct. 30. Disch. Nov. 21. In H. 22 days. *Recovery.* Dr. Garrod, vol. 3, p. 126. Sore throat and pain in swallowing 1 month. Aphonia 1 week. Crepitus at right posterior base. Sputum scanty. Redness of left anterior faucial pillar.

Liq. ammon. acetatis, carbonate of ammonia, and ipecacuanha (13 *days*). *Bark and nitric acid.*

Bronchitis.—A. B., æt. 37, Needlewoman. Adm. Sept. 23. Disch. Oct. 21. In H. 28 days. *Recovery.* Dr. Beale, vol. 6, p. 274. In K. C. H. during Jan., 1863, for 2nd attack of rheum. Prev. ill. 14 days. Œdema of legs. Pain, redness, and swelling of left ankle. Cough and free expectoration. Crepitation over left lung and base of right, elsewhere rhonchus. Systolic bruit loudest at apex. P. 96, R. 40. Slight hepatic enlargement. Tongue furred. Epigastrium tender. Appetite bad.

Sesquichloride of iron, chloric ether, and calumba. Then sp. ammon. arom. and chloric ether.

*Bronchitis—Pleurisy.—Mary H., æt. 20, Servant. Adm. Aug. 6. Disch. Sept. 19. In H. 45 days. *Recovery.* Dr. Johnson. Prev. ill. 6 days. On 7th day cough, pain in chest, brown tongue, bad appetite, crepitation at bases, rhonchus and sibilus over right lung. On 11th day P. 120, more crepitation. On 13th day dulness and feeble breathing over left lower lobe. Tubular breathing at left scapular angle on 15th day. On 18th day brown dry tongue, purged; sores on sacrum, delirious, P. 124, R. 44, urine passed under about this time. Convalescent on 31st day.

Liquor ammon. acetat. (5 *days*). *Sulphate and carbonate of magnesia and chloric ether* (4 *days*). *Liq. ammon. acetat., chloric ether, and sal volatile. Quinine and acid on* 22*nd day. Brandy to* 24 *oz. Turpentine stupes.*

Chronic Bronchitis—Pleurisy.—E. F., æt. 36, Charwoman. Adm. Aug. 5. Disch. Sept. 9. In H. 35 days. *Recovery.* Dr. Garrod, vol. 1, p. 197. Cough every winter. Prev. ill. 1 month. Shivering, dyspnœa, pain in left side. On adm. P. 108, R. 34, expectoration muco-purulent. Dulness diminished, vocal vibration and feeble breathing below left scapular angle; large crepitation over right sup. spinous fossa; elsewhere rhonchus and sibilus. 3 days later large crep. below scapular spines; left base dull. 5 days later br. br. and bronchophony over left lower lobe behind. 9 days later a patch of br. br. at left scapular angle; elsewhere rhonchus and crepitation.

Carbonate of ammonia (4 *days*). *Chloric ether and aromatic spts. of ammonia* (11 *days*). *Senega and carbonate of ammonia. Turpentine stupes.*

Bronchitis and Emphysema.—Elizabeth S., æt. 53. Adm. May 20. *Died* on July 6. In H. 47 days. Dr. Johnson, vol. 15, p. 199. Winter cough 8 years. Prev. ill. 3 weeks. Face extremely livid and congested. Legs slightly œdematous. Mucous rates over chest. Scanty viscid expectoration. P. 120, R. 48. Urine albuminous about 14th day after adm. Legs punctured 14 days later. Copious drain at first, afterwards intense inflammation. Death from exhaustion.

Carbonate of ammonia, chloric ether, and senega. Diuretics. Elaterium. Chlorodyne. Dry cupping. Cucurb. cruent. ad ʒiv. *on 6th day after adm.*

Bronchitis and Emphysema.—J. K., æt. 36, Hatter. Re-adm. Oct. 10. Disch. Nov. 21. In H. 42 days. *Much relieved.* Dr. Beale, vol. 6, p. 302. O. P. since disch. on May 14. Worse about a week; face livid; anasarca; slight ascites; much dyspnœa. Sibilus over lungs above, crepitation at bases, slight dulness at left base. P. 100, R. 28. Systolic bruit over heart. Urine albuminous.

Carbonate of ammonia, chloric ether, and senega. Dilute muriatic acid, chloric ether, and squills. Compound jalap powder. Brandy.

*Bronchitis and Emphysema.—J. H., 49, Clerk. Adm. Nov. 27. *Died* on Dec. 18. In H. 21 days. Dr Garrod, vol. 4, p. 28. Intemperate. Ill 4 months. Pallid and anasarcous; slight ascites; rhonchus and sibilus over lungs; dry cough; P. 104. 4 days later more or less delirious; motions and urine passed under him. On 18th day coarse crepitus all over lungs. P.M.—Much pulmonary emphysema. Left ventricle dilated and hypertrophied. Kidneys wasted and granular. Old clot in left corpus striatum.

Squills, ammonia, and decoction of bark. Iron and quinine. Brandy, 12 *oz.*

Bronchitis and Emphysema.—C. W., æt. 52, Cook. Adm. Sept. 28. Disch. Oct. 17. In H. 19 days. *Recovery.* Dr. Johnson, vol. 17, p. 118. Subject to winter cough. Prev. ill. 5 weeks. Face dusky. Legs œdematous. Slight dry cough. P. 96, R. 28. Rhonchus and sibilus all over chest. Pulsation in scrobiculus.

Chloric ether and carbonate of ammonia. Turpentine stupes.

Bronchitis and Emphysema.—Mary K., æt. 50. Adm. Oct. 14. Disch. Oct. 24. In H. 10 days. *Relieved.* Dr. Johnson, vol. 17, p. 130. Winter cough 12 years. Prev. ill. 6 weeks. Lips blue; legs œdematous; cough and mucous expectoration; universal coarse crepitation.

Carbonate of ammonia, squills, and decoction of broom tops.

Bronchitis and Emphysema.—M. B., æt. 35, Waterman. Adm. Sept. 16. Disch. Oct. 13. In H. 27 days. *Recovery.* Dr. Beale, vol. 6, p. 250. Intemperate. Winter cough 2 years. Has spat blood. Prev. ill. 14 days. Cough and abundant expectoration. Rhonchus and sibilus all over chest. Slight dulness, feeble breathing, and diminished vocal vibration at left base. Tongue dry and furred. P. 108, R. 32.

Carbonate of ammonia and senega. Then carbonate of ammonia, liq. ammon. acetatis and decoct. cinchonæ. Turpentine stupes.

Bronchitis and Emphysema.—Maria S., æt. 38. Adm. Sept. 28. Disch. Oct. 3. In H. 11 days. *Much relieved.* Dr. Garrod, vol. 3, p. 39. Cough and short breath 10 years. Acute rheum, 2 years ago. Face blue, legs œdematous, cardiac dulness increased, rhonchus sibilus and crepitation over lungs, puriform expectoration. P. 96, R. 36. Tenderness over liver.

Carbonate of ammonia, decoction of senega and sp. eth. Nit. Elaterium. Brandy 2 *oz. Turpentine stupes.*

Chronic Bronchitis.—Catherine S., æt. 36. Adm. June 10. Disch. Aug. 12. In H. 63 days. *Unrelieved.* Dr. Beale, vol. 6, p. 186. Ill about 6¼ months. Lately in St. George's. Now much sibilus over chest. Expectoration puriform. P. 60.

Ammonia and chloric ether. Sedatives.

Catarrh.—C. M., æt. 21, Frame Maker. Adm. July 13. Disch. July 24. In H. 11 days. *Recovery.* Dr. Johnson, vol. 17, p. 51. Ill three weeks. Much debility.

Quinine and sesquichloride of iron.

Catarrh.—E. B., æt. 15, Nurserymaid. Adm. July 3. Disch. July 8. In H. 5 days. *Recovery.* Dr. Beale, vol. 6, p. 220.

Liq. ammon. acetatis, sp. ammon. arom., and chloric ether.

Pneumonia.—P. P., æt. 36. Adm. June 24. Disch. July 15. In H. 21 days. *Recovery.* Dr. Johnson, vol. 15, p. 255. Prev. ill. 4 days. Got wet through. Then shivering, cough, dyspnœa and pain in right side. Expectoration rusty on 3rd day (9 days). On adm. skin hot, tongue dry and brown. Dulness and increased vocal vibration over back and upper front of right lung. Bronchial breathing (1 day), over dull space above, small crepn. below. Then returning crepn. Trace of albumen in urine; chlorides scanty. Highest pulse 116, highest resp. 48.

Chloric ether, carbonate of ammonia, and liq. ammonia acetatis (5 *days*). *Then quinine. Brandy* 8 *oz. Turpentine stupes.*

Pneumonia.—Edward G., æt. 7. Adm. Nov. 19. Disch. Dec. 29. In H. 40 days. *Recovery.* Dr. Johnson, vol. 17, p. 157. Ill 3 days. Vomiting. Pain in

right side. Breath short. Tongue furred. P. 144. Acute tenderness of right chest (5 days). Some dulness at right base. Br. br. about scapular angle. On 8th day tubular br. over upper lobe. On 10th day br. br. over scapula. On 12th loud br. br. over right apex. P. 120, R. 30. Br. everywhere vesicular on 33rd day.

Liq. ammon. acetatis, ipecacuanha, sp. eth. nit., and liq. morph. mur. (8 *days*). *Then quinine. Wine* 8 *oz. Cod liver oil. Sinapism to back.*

Pneumonia.—Harry T., æt. 10. Adm. June 15. Disch. July 11. In H. 26 days. *Recovery.* Dr. Garrod, vol. 2, p. 182. Ill 1 day, with shivering, cough, loss of appetite and pain in left side. Now flushed, left chest painful and tender on pressure (4 days). Dulness, loud bronchial br., and bronchophony below spine of left scapula (14 days). No expectoration. Highest pulse 144, highest resp. 84.

Chloric ether, carbonate of ammonia and liquor ammoniæ acetatis. Afterwards quinine.

Pneumonia-Phlegmasia dolens.—C. L., æt. 24, Waiter. Adm. Aug. 7. Disch. Sept. 26. In H. 50 days. *Recovery.* Dr. Beale, vol. 5, p. 256. Slight purpura 3 weeks. Phlegmasia dolens of right leg 2 weeks. Pain in right chest on day after adm., hœmoptysis next day; 2 days later dulness over lower lobes of lungs, small crep. over right, large crep. over left, sputa bloody, P. 100, R. 36; 3 days later loud pleuritic rub over right lower lobe; 4 days later right leg larger, fresh petechiæ; 5 days later phleg. dolens of left leg.

Liq. cinchonæ and citrate of potass. Dilute muriatic acid and bark. Belladonna ointment to legs.

Pneumonia.—J. S., æt. 38, Sign Writer. Adm. July 2. Disch. Sept. 19. In H. 79 days. *Recovery.* Dr. Beale, vol. 5, p. 216. Prev. ill. 5 days. Slight hœmoptysis, recurring on morning of adm. Pain in left side. Now P. 72, slight dulness and feeble br. over whole left lung. Four days later, increased dulness and diminished vocal vibration over left side, behind, bronchial br. below angle of scapula, expectoration viscid and rusty. Next day large crepn., mixed with br. br. below scap. angle. P. rose to 106. On 67th day friction sound over left base. On 75th day br. vesicular, rub still audible.

Gallic acid and opium. Then sal volatile, chloric, ether and liq. ammon. acetatis. Nitric acid and bark. Compound iodine and mercurial ointment applied over lungs.

Pneumonia.—E. W., æt. 20, Salesman. Adm. Oct. 23. Disch. Nov. 16. In H. 24 days. *Recovery.* Dr. Beale, vol. 5, p. 326. Prev. ill. 5 days. Shivering, cough, loss of appetite, slight hœmoptysis. Dulness, fine crepitation and increased vocal vibration over right lower lobe below scapular angle, up to spine 4 days later; sputa rusty at first; now viscid and colourless. Highest pulse 84, highest resp. 48.

Sp. ammon. arom., and liquor ammoniæ acetatis. Dilute muriatic acid and quassia. Brandy 6 *oz. Turpentine stupes. E. L. to right side.*

Pneumonia.—Mary A. C., æt. 14. Adm. July 14. Disch. Aug. 12. In H. 29 days. *Recovery.* Dr. Johnson, vol. 18, p. 1. Ill 2 days. Shivering, headache, thirst, anorexia. Tongue furred. Dry cough. Much pain and tenderness on pressure, slight dulness, and harsh bronchial expiration under right clavicle. Some dulness at right base. P. 124. R. 64.

Chloric ether and liquor ammon. acetatis. Then quinine and iron. Aperients.

Pleuro-Pneumonia.—H. W., æt. 19, Compositor. Adm. Sept. 26. Disch. Nov. 7. In H. 42 days. *Recovery.* Dr. Johnson, vol. 19, p. 88. Prev. ill. 6 days. Pain in left side, dyspnœa, cough, hœmoptysis. On adm. dulness, bronchial

breathing, œgophony, friction sound, and diminished vocal vibration, below left scapula. Expectoration viscid and rusty.

Liquor ammonia acetatis (9 *days*). *Then sesquichloride of iron and quinine. E. L. to left side on* 16*th day.*

Pleurisy.—G. E., æt. 37, Carpenter. Adm. July 1. Disch. Sept. 14. In H. 75 days. *Recovery.* Dr. Johnson, vol. 16, p. 263. Prev. ill. 6 weeks. Began with pain in right side. On adm. P. 100, R. 32, right side dull all over behind, and below 3rd rib in front (up to clavicle on 7th day after adm.). Absence of breathing in front; œgophony and faint tubular breathing below angle of scapula behind; vocal vibration diminished. 42 days later breathing everywhere vesicular.

Iodide of potassium, chloric ether and bark (14 *days*). *Then quinine and acid. Ol. Morrhuæ. Iodine paint to right side.*

Pleurisy.—M. G., æt. 35, Pavior. Adm. June 11. Disch. July 1. In H. 20 days. *Relieved.* Dr. Beale, vol. 6, p. 182. Pain in left side 3 weeks. On adm. dulness, diminished vocal vibration, and absence of breathing below 3rd rib in front, and spine of left scapula. Pleuritic rub above 3rd rib. P. 120, R. 38. 15 days later loud friction sound, and feeble breathing all over left side.

Liq. ammoniæ acetatis, carbonate of ammonia, chloric ether and broom tops.

Pleurisy.—Sam. B., æt. 30, Singer. Adm. Nov. 13. Disch. Dec. 15. In H. 32 days. *Recovery.* Dr. Johnson, vol. 19, p. 152. Prev. ill. 3 days. Stabbing pain in left side. Dulness with absence of breathing, and vocal vibration below 3rd rib in front and angle of left scapula. Pleuritic rub below left nipple. Œgophony. Sputa, viscid, untinged. On 11th day coarse crep. at left post. base (21 days). Much pain in side during recovery.

Aromatic spts. of ammonia, liq. ammon. acetatis, and bark (4 *days*). *Then quinine. Iodine paint to side.*

Pleurisy.—W. S., æt. 28, Carrier. Adm. Oct. 16. Disch. Dec. 9. In H. 54 days. *Recovery.* Dr. Johnson, vol. 18, p. 43. Prev. ill. 21 days. Dyspnœa and pain in right side Œdema of legs 15 days later. On adm. dulness below 5th rib, in front, and right scapular spine. Friction sound near nipple. Breathing distant above, bronchial at angle, absent at base. Sputum clear. P. 78, R. 30. 31 days later flattening of right chest and sinking of right shoulder.

Liq. ammoniæ acetatis, chloric ether and aromatic spts. of ammonia.

Pleurisy.—H. S., æt. 23, Labourer. Adm. Oct. 1. Disch. Nov. 7. In H. 37 days. *Recovery.* Dr. Beale, vol. 5, p. 308. Prev. ill. 12 days. Pain in right side, dry cough, dyspnœa. Dulness, absence of breathing, and vocal vibration below upper margin of right scapula, and below nipple. P. 96, R. 40. Friction sound below right scapular angle on 17th day (20 days), and bronchial breathing at scapula angle about 24th day.

Liquor ammoniæ acetatis, sp. ammon. arom., and chloric ether. E. L. to right side on 31*st day.*

Pleurisy.—Mary H., æt. 20, Servant. Adm. June 24. Disch. July 18. In H. 24 days. *Recovery.* Dr. Johnson, vol. 15, p. 261. Prev. ill. 3 weeks. Pain in left side, dry cough. Dulness below 4th rib, in front, and centre of left scapula. Breathing absent at base, feeble elsewhere. Slight œgophony. Five days after adm. pleuritic rub at base, audible till discharge.

Liquor ammoniæ acetatis and chloric ether (14 *days*). *Then quinine and iron. Aperients.*

Pleurisy.—Sarah B., æt. 35, Servant. Adm. July 6. Disch. Aug. 12. In H. 37 days. *Recovery.* Dr. Beale, vol. 5, p. 220. Prev. ill. 3 days. Abdominal and interscapular pain, vomiting, shivering, and loss of appetite. On adm. tongue furred, P. 120, R. 24. Next day pleuritic rub (22 days) at right scapular angle. Later, dulness and absence of breathing at base. Liver enlarged.

Liq. ammoniæ acetatis, chloric ether and aromatic spts. of ammonia. Then

dilute nitric acid and bark. Taraxacum. Purgatives. Hydrochloric acid lotion.

Pleurisy.—F. N., æt. 20, Sempstress. Adm. June 17. Disch. July 1. In H. 14 days. *Recovery.* Dr. Beale, vol. 6, p. 192. Prev. ill. 7 days. Pain in right side, dry cough, thirst, loss of appetite. On adm. P. 108, R. 40. Slight dulness, feeble breathing, and diminished vocal vibration below right scapular angle; friction sound two days later.

Liquor ammon. acetatis (5 *days*). *Then iron and quinine. E. L. to side.*

Pleurisy.—Eliza B., æt. 20, Servant. Adm. Nov. 14. Disch. Dec. 2. In H. 11 days. *Recovery.* Dr. Beale, vol. 6, p. 323. Pain in right side 6 days. On adm. friction sound near angle of right scapula (5 days). P. 92. Probably tubercular.

Liq. ammon. acetatis and sal volatile (10 *days*). *Quinine. Brandy. Aperients.*

Empyema.—Edwin H., æt. 13. Adm. Aug. 24. Disch. Oct. 7. In H. 44 days. *Relieved.* Dr. Garrod, vol. 2, p. 261. Prev. ill. 8 months. At first febrile symptoms and pain in left side. Five months later a tender red tumour between left 4th and 5th ribs 2 in. below nipple, and 1½ in. to left of sternum; opened 6 weeks ago. Now pallid, left shoulder sunk, side flattened, girth at nipple 2 in. less than right side, about ℥iii. pus daily from opening. Left side dull except above 3rd rib in front. Vocal tremor absent. Breathing vesicular to mid scapula, below this to base bronchial with ægophony, harsh and crackling under clavicle. P. 88. Skin hot and dry. Discharge fell to ℥½ daily.

Nitric acid and bark. Afterwards cod liver oil and syrup of iodide of iron. Left side tightly strapped below opening.

Phthisis.—J. E., æt. 81. Adm. June 16. *Died* on July 31. In H. 45 days. Dr. Garrod, vol. 2, p. 174. Cough 2 years. Worse 4 days ago. Right upper lobe dull on percussion, cavernous breathing, gurgling and pectoriloquy under clavicle, pectoriloquy at post. apex. P. 96. P.M. Right lung adherent, 1 pint fluid in base of pleura. Right lung stuffed with tubercles; vomica in post. apex. Slight tubercular deposit in left apex.

Carbonate of ammonia and bark. Cod liver oil. Dilute sulphuric acid and bark.

Phthisis.—T. C., æt. 47, Silver Polisher. Adm. Nov. 10. *Died* on Nov. 15. In H. 5 days. Dr. Johnson, vol. 19, p. 148. Cough 3 winters. Loss of flesh 6 months. Hoarse 2½ months. Dulness, amphoric breathing, and cracked pot sound under left clavicle; dulness, br. br., and crep. over left back. Tubular br. and crep. under right clavicle. P. 120. P.M.—Large cavern in left upper lobe. Much tubercle elsewhere in lungs. Liver large.

Sp. ammon. arom., hyoscyamus and bark. Syrup of iodide of iron and cod liver oil.

Phthisis.—J. M., æt. 27, Coachman. Adm. Sept. 20. *Died* on Oct. 30. In H. 40 days. Dr. Garrod, vol. 3, p. 21. Hereditary. Cough and loss of flesh, 5 months. Diarrhœa. Hæmoptysis at times, 6 weeks. 3 pts. blood on day before adm. Dulness and fine crep. at left apex; gurgling and tubular br. under right clavicle; crep. at post. apex; slight dulness and fine crep. over right back; P. 120, R. 40. Sputa nummular streaked with blood; 1½ pints daily twenty days later.

Dilute phosphoric acid, glycerine, morphia, and gentian. Syrup of iodide of iron and cod liver oil. Liq. ammon. acet., digitalis, and bark. I. P. to right chest.

Phthisis.—W. B., æt. 49, Compositor. Adm. July 27. Disch. Aug. 8. In H. 12 days. *Unrelieved.* Dr. Johnson, vol. 19, p. 34. Always subject to cough and expectoration. Very anæmic. Dulness under left clavicle; large crep. to nipple;

crep. and prolonged expectoration at left post. apex ; crep. under right clavicle. Puriform sputa. P. 100. Night sweats. Liver large and tender.

Cod liver oil. Dilute muriatic acid, chloric ether, and quinine.

Phthisis.—Cath. C., æt. 13. Adm. July 29. Disch. Aug. 5. In H. 7 days. Dr. Beale. Cough and loss of flesh 5 months. Hæmoptysis on evening of adm. and 4 days ago. Slight dulness over left upper lobe ; loose crep. and tubular br. under clavicle ; smaller crep. below. Harsh br. and crackling under right clavicle. Sputa nummular.

T. opii, dilute sulphuric acid, and chloric ether. Cod liver oil. E. L. under left clavicle.

Phthisis.—Robert E., æt. 24. Adm. Sept. 26. Disch. Oct. 31. In H. 35 days. *Very much relieved.* Dr. Beale, vol. 5, p. 298. Had phthisis 6 years. Worse 3 months. Vomited blood 2 years ago. Frequent hæmoptysis. Left lung dull back and front. Gurgling tubular br. and pectoriloquy under clavicle ; tubular br. in lower lateral region. Breathing and vocal vibration absent at base. Spleen much enlarged (2 years). Expectoration viscid, muco-purulent, somewhat rusty. Appetite good.

Quinine. Pepsine. Cod liver oil. Iodine paint to left chest.

Phthisis.—H. L., æt. 28, Cook. Adm. Oct. 28. Disch. Dec. 23. In H. 56 days. *Relieved.* Dr. Beale, vol. 5, p. 330. Ill 8 months. Pain in chest ; cough. Dulness, and 5 days later br. br. and pectoriloquy under right clavicle ; rhonchus and sibilus over rest of lung ; P. 128, R. 40. 23 days later coarse crep. under right clavicle ; pain under left.

Sesquichloride of iron. Bismuth. Cod liver oil. Blisters under right clavicle.

Phthisis.—C. F., æt. 27, Compositor. Adm. Sept. 11. Disch. Sept. 23. In H. 12 days. *Relieved.* Dr. Beale, vol. 5, p. 286. Prev. ill. 7 months. Cough. Pain in right side. Night sweats. Loss of flesh. Slight hæmoptysis. Flattening, dulness, crepitation, and prolonged expectoration under right clavicle. Crepitation over sup. spinous fossa. Expectoration scanty.

Cod liver oil. Aromatic sulphuric acid, paregoric and bark.

Phthisis.—Emma M., æt. 15. Adm. Sept. 26. Disch. Oct. 17. In H. 21 days. *Much relieved.* Dr. Beale, vol. 6, p. 294. Weakness and emaciation 6 weeks. Cough 14 days. Dulness, cracked pot sound, large crepitation, and increased vocal resonance below left clavicle. Fine crep. over left back ; bronchial br. 9 days later.

Liq. ammon. acetatis. Syrup of iodide of iron and cod liver oil. Iron and calumba.

Phthisis.—Elizabeth T., æt. 21, Works Sewing Machine. Adm. July 14. Disch. Aug. 29. In H. 46 days. *Unrelieved.* Dr. Garrod, vol. 1, p. 175. Hereditary. Subject to cough. Hæmoptysis 16 months ago. Loss of flesh 9 months. Night sweats. Chest resonant ; crepitation over lower front and back of lungs. Appetite bad.

Sulphuric acid and opium. Muriate of ammonia. Bismuth. Tannic acid and conium.

Phthisis.—E. L., æt. 44, Needlewoman. Adm. Sept. 15. Disch. Sept. 26. In H. 11 days. *Relieved.* Dr. Johnson. Ill 2 years. Cough and expectoration. Loss of flesh. Hæmoptysis (½ pt.) 14 days ago and since. Dulness, prolonged expiration, loose crepitation, and bronchophony over left apex. P. 104, R. 40.

Turpentine inhalation. Sulphate of iron and sulphuric acid. Quinine. Cod liver oil and syrup of iodide of iron.

Phthisis.—W. S., æt. 29, Butcher. Adm. Sept. 21. *Died* on Sept. 25. In H. 4 days. Dr. Beale, vol. 6, p. 264. Cough, pain in right chest, and emaciation, 12 months. Worse 8 days. Now, orthopnœa, sputa rusty. P. 124, R. 50. Gurgling and tubular br. under right clavicle ; right lung very dull at back ; br.

br. below mid-scapula; gurgling and tinkling at angle; puerile br. over lower part of right lung in front: elsewhere crepitus.

Carbonate of ammonia and senega. Ether. Brandy. Turpentine stupes.

PHTHISIS.—W. B., æt. 24, Waiter. Adm. Dec. 20. *Died* on Dec. 29. In H. 9 days. Dr. Garrod. Cough, short breath, and loss of flesh, 15 months. 6 days ago profuse hæmoptysis; occasional attacks during last 8 months. Now very pallid. Dulness and diminished expansion under right clavicle; crepitation over right lung and left sup. spinous fossa. Expectoration purulent, very bloody. Night sweats. P. 100, R. 36. Fresh hæmoptysis, followed by exhaustion and death. P.M.—Cavity size of a small orange in right apex filled with clotted blood; left apex solid, with tubercle; elsewhere scattered tubercles. Emphysema.

Gallic and sulphuric acid. Quinine and sulphate of iron. Turpentine. Turp. inhalation.

PHTHISIS—SECONDARY SYPHILIS.—Rose H., æt. 29. Adm. May 11. Disch. May 18. In H. 7 days. Dr. Beale, vol. 5, p. 160. Hæmoptysis 3 years ago; cough since. Loss of flesh. Dulness and prolonged harsh expiration under right clavicle. Vagina ulcerated into rectum. Vesico vaginal fistula. Transferred to surgical ward on May 18.

HÆMOPTYSIS.—M. C., æt. 42, Saleswoman. Adm. July 31. Disch. Aug. 15. In H. 15 days. *Unrelieved.* Dr. Garrod, vol. 1, p. 191. Weak and thin 6 months. Cough 1 month. Night sweats. Hæmoptysis (3 pts.) on morning of adm. Harsh breathing under clavicles. No dulness. P. 68.

Tannic acid, dilute sulphuric acid, and ipecacuanha. Morphia.

HÆMOPTYSIS.—J. H., æt. 59, Hawker. Adm. Dec. 7. Disch. Dec. 30. In H. 23 days. *Recovery.* Dr. Johnson, vol. 18, p. 129. Cough lately, but no other symptoms of phth. Hæmoptysis on 6th and 7th, and again 3 days later. Harsh br. over right post. apex. Some crepitation at bases.

Gallic acid. Turpentine. Iron. Turpentine inhalation.

ASTHMA.—Eliza K., æt. 34, Married. Adm. June 29. Disch. July 11. In H. 12 days. *Recovery.* Dr. Garrod. Cough and expectoration 1 month. Nightly attacks of dyspnœa and spasmodic cough, relieved by expectoration (14 days).

Sulphuric ether, lobelia, and quinine.

ASTHMA.—Caroline H., æt. 21. Adm. Oct. 17. Disch. Oct. 24. *Relieved.* Dr. Johnson, vol. 18, p. 59. Hereditary. Always subject to attacks of dyspnœa and cough; now has 2 or 3 daily. Worse in winter. Has emphysema.

Sulphuric ether, lobelia, syrup of iodide of iron, and cod liver oil.

PERTUSIS—PNEUMONIA.—Henry T., æt. 8. Adm. Nov. 13. Disch. Dec. 23. In H. 40 days. *Convalescence.* Dr. Garrod, vol. 3, p. 140. Strumous. Cough 2 years, lately whooping. Scanty purulent expectoration. Back of left lung quite dull, breathing absent below, bronchial above, in front vesicular with rhonchus. P. 116, R. 36.

Cod liver oil and syrup of iodide of iron. Belladonna. Conium.

PERTUSIS—PNEUMONIA.—Ellen M., æt. 10. Adm. July 14. Disch. Oct. 17. In H. 95 days. *Much relieved.* Dr. Beale, vol. 5, p. 244. Whooping cough on adm. 20 days later spat up o.ss of blood. Next day dulness br. br. and crepitation below left scapular angle, with diminished vocal vibration 7 days later. Sputa viscid and tinged with blood. 56 days afterwards br. br. at right scapular angle. On discharge dulness about angle of left and below spine of right scapula. Breathing bronchial over left side, and bronchial with coarse crep. over left back.

Sp. ammon. arom., chloric ether and liq. ammon. acetatis. Syrup of iodide of iron and cod liver oil. Turpentine stupes, and ung. iodinii co. to back.

Acute Laryngitis.—Louisa W., æt. 41, married. Adm. June 8. Disch Aug. 1. In H. 54 days. *Recovery.* Dr. Johnson, vol. 15, p. 231. Syph. sore 2 years ago. Aphonia (6 months) 1½ years ago. Now hoarse 6 months. Dysphagia 1 month. Psoriasis on knees and elbows. On adm. face dusky, much dyspnœa and laryngeal stridor. Glottis reduced by swelling to a small triangular chink posteriorly. Tracheotomy next day. Patient did well; tube removed after 36 days, and wound almost well 5 days later. Slight ulceration of inter-arytenoid fold.

Iodide of potassium. Calomel and colocynth. Leeches over larynx. Mercurialized afterwards. Quinine and iron.

Laryngitis.—Jane E., æt. 32, Hair Net Maker. Adm. Nov. 11. Disch. Dec. 19. *Convalescent.* In H. 38 days. Dr. Johnson, vol. 17, p. 165. O. P. since disch. on Ap. 22, convalescent from laryngitis. Huskiness and, at times, aphonia. Larynx red and congested. Voice natural on 33rd day.

Liq. ammon. acetatis, henbane, chlorate of potass and chloric ether. Quinine and iron. Steam inhalation. Belladonna ointment externally.

Croup.—David M. æt. 3. Adm. Dec. 5. *Died* on Dec. 5. Dr. Beale, vol. 8, p. 8. Delicate. Attacked on morning of adm. Great dyspnœa and exhaustion, cough not brassy, no stridor. Died, exhausted, about 3 hours after adm.

Ipecacuhana emetics. Belladonna and bicarbonate of potass. Belladonna ointment to front, and sinapism to back of neck.

Croup.—Frederick C., æt. 3. Adm. Aug. 24. *Died* on Aug. 25. Dr. Johnson, vol. 17, p. 86. Catarrh and sore throat 19 days. Dyspnœa 4 days. Had treatment. On adm. much dyspnœa, face dusky, P. small and rapid, short cough, crepitation at bases of lungs. Temporary relief from tracheotomy; increase of dyspnœa; exhaustion; death.

Tracheotomy. Brandy and egg. Vin. ipecac.

Aphonia (incomplete).—Eliza K., æt. 20, Servant. Adm. May 20. Disch. July 1. In H. 40 days. *Improved.* Dr. Johnson. vol. 17, p. 43. Weakness and loss of flesh 5 months. Voice husky 6 weeks. Slight cough. Laryngeal mucous membrane anæmic.

Quinine and iron. Ipecac. and squill pill.

Laryngeal Ulceration.—Bridget M., æt. 22, Servant. Adm. June 27. Disch. July 22. In H. 25 days. *Unrelieved.* Dr. Johnson, vol. 16, p. 259. Cough 8 months. Hoarse 5 months. Can only speak in a whisper. Signs of softening tubercle at right apex.

Iodide of potassium and liquor cinchonæ. Chlorate of potass gargle. Hirud. i *over larynx.*

Laryngeal Ulceration.—Ann M. æt. 30, Dressmaker. Adm. July 23. Disch. Aug. 19. In H. 27 days. *Unrelieved.* Dr. Johnson. Had syphilis. Ill 6 weeks. Aphonia. Glottis congested. Ulceration of left false cord.

Quinine and iron. Cod liver oil. Steam inhalation. Sinapisms to throat.

Laryngeal Ulceration.—Anne E., æt. 32. Adm. June 17. Disch. Aug. 29. In H. 73 days. *Unrelieved.* Dr. Johnson, vol. 16, p. 251. Cough 7 months. Hoarse 3 weeks. Dysphagia and sore throat 10 days. Now complete aphonia, ulcer on right edge of thick red epiglottis, and on right false cord. Swelling of false cords and inter-arytenoidal fold. Tubercle at right apex.

Quinine and sulphuric acid. Cod liver oil. Sol. A. N. to larynx.

Epithelioma of Epiglottis.—William H., æt. 64, Street News Seller. Adm. Sept. 19. Disch. Oct. 14. In H. 25 days. *Unrelieved.* Dr. Johnson. Became

worse after discharge on May 27. On Aug. 10 coughed up an epitheliomatous mass as large as an almond. Voice and deglutition better since.

Chlorate of potass and sesquichloride of iron.

Was re-admitted on Jan. 12, 1864. On Feb. 6 was discharged at his own request. Re-adm. on April 19. The tumour was then much ulcerated on the surface. *Died* suddenly on July 23rd. P.M.—Epiglottis converted into a firm, nodulated epitheliomatous mass, the size of a small apple. The upper part of the left arytenoid cartilage had become atrophied from pressure. The mass was impacted in the glottis. Larynx and tongue healthy. No cancer elsewhere.

Suffocation.—W. D., æt. 15 months. Brought in dead on June 2. Dr. Johnson. No marks of violence. When brought in, trunk warm. P.M.—Slight congestion of cerebral blood-vessels. Verdict on inquest "found dead."

Sore Throat.—S. W., æt. 16, Servant. Adm. May 28. Disch. June 13. In H. 16 days. *Recovery.* Dr. Johnson, vol. 17, p. 17. Prev. ill. 3 days. High fever, yellowish exudation on tonsils. Convalescent on 11th day.

Chlorate of potass, dilute muriatic acid and bark (9 days). Then quinine and sesquichloride of iron. Glycerine and iron to fauces.

Cynanche Tonsillaris.—P. C. æt. 30, Bootmaker. Adm. June 11. Disch. June 17. In H. 6 days. *Recovery.* Dr. Garrod, vol. 2, p. 162. First attack. Prev. ill. 7 days. Right tonsil much swelled. Ulcer on left of uvula. Great dysphagia. Convalescent on 11th day.

Liquor ammoniæ acetatis, and mist. alba o. m. (4 days.) Then nitro-muriatic acid and bark.

Cynanche Tonsillaris.—S. S., æt. 30, Hospital Nurse. Adm. Nov. 9. Disch. Dec. 5. In H. 26 days. *Recovery.* Dr. Beale, vol. 6. p. 330. Prev. ill. 3 days. Abscess in left tonsil.

Liq. ammon. acetatis, ether and bark. Dilute muriatic acid and liq. cinchonæ. Wine 4 oz. Iron and glycerine to throat.

Cynanche Tonsillaris.—E. C., æt. 20, Housemaid. Adm. Nov. 18. Disch. Nov. 25. In H. 7 days. *Recovery.* Dr. Garrod, vol. 1, p. 236. Prev. ill. 2 days. Both tonsils swelled. Abscess in left tonsil, and whitish exudation on its surface.

Compound tinct. of lavender and mist. camph. Iron and glycerine to throat.

Ulceration of Throat.—G. C., æt. 24, Shopman. Adm. Aug. 11. Disch. Aug. 27. In H. 16 days. *Recovery.* Dr. Johnson. No syphilis. Prev. ill. 4 days. Chilled while sweating. On adm. tonsils and uvula red and swollen, sloughing of left tonsil and left side of uvula, ulceration of right tonsil. 2 days later slough on post. wall of pharynx; slight delirium.

Carbonate of ammonia, chloric ether and bark. Brandy 9 oz. Afterwards quinine.

Syphilitic Ulceration of Throat.—S. B., æt. 31, Labourer. Adm. June 19. Disch. July 27. In H. 38 days. *Recovery.* Dr. Johnson, vol. 16, p. 249. Sore throat last March. Has lived badly since; discharge from K.C.H. Prev. ill. 6 weeks. Excavated ulcers of tonsils, deepest on left tonsil; back of pharynx ulcerated. Bones of right arm tender and painful.

Chlorate of potass and sesquichloride of iron. Iodide of potassium, liquor cinchonæ, and muriate of morphia. Local application of nitrate of silver.

Syphilitic (?) Ulceration of Throat.—C. P., æt., 25, Wood Carver. Adm. June 6. Disch. July 1. In H. 25 days. *Recovery.* Dr. Johnson, vol. 16, p. 241. Intemperate. Chancre 2 months ago. Sore throat since. Tonsils red, swollen, and ulcerated on surface.

Chlorate of potass and bark (6 days). Then chlorate of potass and sesquichloride of iron.

Ulceration of Throat.—Matilda M., æt. 45, Married. Adm. July 17. Disch. Sept. 2. In H. 47 days. *Recovery.* Dr. Johnson, vol. 19, p. 20. Sore throat 2 months. Aspect cachectic. Uvula and most of soft palate destroyed by ulceration. Left side of throat sloughing.

Iodide of potassium, sesquichloride of iron, and morphia. Belladonna. Local application of nitric acid.

Mercurial Salivation—Stomatitis.—T. S., æt. 27, House Painter. Adm. July 4. Disch. July 18. In H. 14 days. *Nearly well.* Dr. Johnson, vol. 17, p. 177. Never suffered from lead. Subject to gout. Swelling in right groin 3 weeks ago; ten days ago took some pills; 3 days later mouth sore, much salivation. On adm. lower part of face swelled; gums spongy and bleeding; tongue swollen, cannot be protruded far; breath fœtid.

Chlorate of potass, liq. cinchonæ, and chloric ether. Chlorine gargle.

Stricture of Œsophagus.—M. B. æt. 33, Cook. Adm. Oct. 17. Disch. Nov. 16. In H. 30 days. *Relieved.* Dr. Johnson, vol. 17, p. 132. Dysphagia 7 or 8 months, worse last month. Emaciation 1 month. Hoarseness and pain in throat 14 days.

Hot fomentations and steam inhalations. Brandy and beef tea enemata. Afterwards sedatives. Stricture:—dilated by catheters (No. 9–12).

Cancer of Œsophagus—General Paralysis.—H. D., æt. 52, Porter. Adm. July 18. *Died* on Aug. 20. Dr. Johnson, vol. 18, p. 5. Intemperate. Syphilis 35 years ago. 3 months ago loss of power in arms and legs, numbness of hands and feet, headache, occasional vertigo, and temporary unconsciousness. On adm. unable to walk without help; cannot pick up a pin. Radials rigid. P. 56. Marked arcus senilis. 9 days later incontinence of urine. 8 days later dysphagia. 11 days later died suddenly of asphyxia. P.M.—Pericardium universally adherent. Heart fatty. Medullary cancer of œsophagus, forming a tumour the size of an egg, chiefly between œsophagus and trachea; an opening from mass into trachea near bifurcation, through which creamy fluid flowed into trachea. Subarachnoid fluid increased. Brain healthy. Cord not examined.

Iodide of potassium and bark. Strychnia.

Dyspepsia.—W. B., æt. 41, Commissionaire. Adm. Sept. 23. Disch. Oct. 2. *Recovery.* Dr. Johnson, vol. 17, p. 102. Had dysentery in India. Pain and soreness at epigastrium and vomiting 12 days. Coffee-ground matters in vomit. Furred tongue. Bowels confined.

Bismuth. Ammonio-citrate of iron.

Dyspepsia.—R. R., æt. 53, Shopman. Adm. Sept. 29. Disch. Oct. 21. In H. 22 days. *Recovery.* Dr. Johnson, vol. 19, p. 95. Hæmatemesis 8 and 7 years, Jaundice, 2 years ago. Epigastric pain and tenderness, and constipation 2 months. Vomiting 6 weeks, becoming fœcal during last 4 or 5 days. Bowels confined 7 days. Very weak. Epigastrium and left hypochondrium tender. Sarcinæ in vomit.

Enemata of castor oil and warm water. Bismuth and hydrocyanic acid. Oxide of silver and henbane. Sinapisms to epigastrium.

Dyspepsia.—W. H., æt. 45, Labourer. Adm. Oct. 19. Disch. Oct. 24. In H. 5 days. *Relieved.* Dr. Garrod, vol. 3, p. 90. Occasional vomiting after meals, 4 years. Sudden pain at epigastrium 2 years ago while lifting a heavy

weight. Ten weeks afterwards hæmatemesis, melæna, and return of pain. Epigastric pain 1 week. Vomiting 5 days. Tongue white.

Bismuth and calumba.

Dyspepsia.—Selina P., æt. 48, Servant. Adm. June 10. Disch. July 8. In H. 28 days. *Nearly well.* Dr. Beale, vol. 5, p. 196. Pain and soreness of epigastrium, 2 months. Waterbrash and loss of appetite, flesh, and strength, 1 month. Vomiting; anæmia.

Bismuth and dilute hydrocyanic acid. Henbane, sesquichloride of iron, and dilute hydrocyanic acid.

Dyspepsia.—F. M., æt. 61, Monthly Nurse. Adm. Sept. 26. Disch. Oct. 28. In H. 32 days. *Relieved.* Dr. Beale, vol. 6, p. 280. Pain across epigastrium, vomiting, and hæmatemesis 2½ months ago. Jaundice 14 days later. Loss of appetite, sour risings, waterbrash, constipation. Moveable painless tumour in left hypochondrium, probably connected with left lobe of liver.

Mist. Rhei. Co.

Dyspepsia—Partial Peritonitis?—H. G., æt. 44, Probationer, K.C.H. Adm. July 12. Disch. Sept. 25. In H. 75 days. *Recovery.* Dr. Johnson, vol. 16, p. 269. Pain after food and waterbrash 6 weeks. On day before adm. great pain in abdomen and vomiting. Some blood in vomit. Epigastrium extremely tender. Tongue furred. Appetite bad. P. 96. Free from acute symptoms 8 days later. Relapse 16 days later.

Bismuth hydrocyanic acid and muriate of morphia. Citrate of iron, hydrocyanic acid, and calumba. Iron and compound galbanum pill.

Hæmatemesis.—Ellen C., æt. 28, Married. Adm. Nov. 23. Disch. Dec. 16. In H. 23 days. *Recovery.* Dr. Garrod, vol. 19, p. 180. Not subject to vomiting or abdominal pain. On day of admission, soon after lifting a tub of water, pain in left side, faintness and copious hæmatemesis. Hæmatemesis recurred once on night of 24th, twice on 25th, and once on night of 25th. Much blood passed by bowels during this time.

Gallic acid. Ice. Enemata of brandy and beef tea. Turpentine. Sesquichloride of iron and quinine.

Hæmatemesis.—L. A., æt. 47, Cook. Adm. Sept. 18. Disch. Sept. 26. In H. 18 days. *Recovery.* Dr. Johnson, vol. 19, p. 97. Intemperate. Four days ago, faintness followed by vomiting of blood. No dyspeptic symptoms or fresh hæmatemesis.

Turpentine and chloric ether. Afterwards iron and columba.

Pyloric Obstruction.—A. B. S. R., æt. 69. Adm. Sept. 23. Disch. Oct. 5. In H. 12 days. *Very much relieved.* Dr. Beale, vol. 5, p. 290. Father died of cancer. During last month pain and soreness of epigastrium, much flatulence, vomiting, and loss of flesh. Appetite bad. Stomach much dilated. Vomit contains sarcinæ. Urine 1-12th albumen. No tumour.

Bismuth and paregoric. Creosote.

Pyloric Obstruction.—J. C., æt. 49, Spurmaker. Adm. Nov. 3. Disch. Nov. 18. In H. 15 days. *Much relieved.* Dr. Johnson, vol. 17, p. 141. Formerly drank large quantities of beer. Vomiting, loss of appetite, flesh, and strength, 18 months. Has vomited twice or thrice daily for last six weeks. Very pale and thin. Stomach enormously dilated. Vomit contains sarcinæ. No tumour.

Strychnia and dilute muriatic acid. Strychnia and sesquichloride of iron. Purgatives.

Cancer of Stomach.—D. H., æt. 47, Cellarman. Adm. June 11, again Sept. 24 and Nov. 7. *Died* on Nov. 11. Dr. Johnson, vol. 17, p. 87. Loss of flesh

and strength, and pain at epigastrium after food, 2 months. Loss of appetite, 6 weeks. On adm. a hard moveable tumour the size of a large orange in umbilical region of abdomen. Occasional vomiting. In H. 37 days. On re-adm., Sept. 24, more emaciated; tumour larger and more tender. In H. 50 days. Re-admitted on Nov. 7. Abdomen tympanitic; vomiting daily. Four days later severe abdominal pain, followed by collapse and *death*. P.M. Perforation of ascending colon. Recent peritonitis; cancer of stomach, involving colon and part of small intestine, but not stricturing pylorus.

Quinine. Iron and hydrocyanic acid. Bismuth. Opium.

JAUNDICE.—A. P., æt. 32, Blacksmith. Adm. Nov. 9. Disch. Dec. 16. In H. 37 days. *Recovery.* Dr. Beale, vol. 5, p. 349. Subacute rheumatism 4 months ago. Urticaria 14 days ago. Jaundice 10 days. Lemon coloured, languid, sleeps badly. Motions clay coloured. No hepatic pain, tenderness, or enlargement. Right hip and shoulder tender.

Iodide of potassium, bicarbonate of potass, and decoction of bark (12 *days*). *Then dilute muriatic acid and decoction of bark. Hydrochloric acid lotion. Grey powder and rhubarb.*

JAUNDICE.—Frances W., æt. 34, Married. Adm. July 17. Disch. Aug. 1. In H. 15 days. *Recovery.* Dr. Garrod, vol. 2, p. 232. Five months pregnant. Ill 8 days. Nausea, loss of appetite, jaundice. Motions white. No hepatic pain, tumour, or tenderness.

Castor oil. Quinine and sulphuric acid.

JAUNDICE.—E. B., æt. 20, Servant. Adm. Sept. 7. Disch. Sept. 23. In H. 16 days. *Recovery.* Dr. Garrod, vol. 2, p. 271. Jaundice, vomiting, and constipation, 1 week. Skin deep yellow; pain and tenderness across upper part of abdomen. Liver not enlarged. Motions white. Soft systolic bruit at base of heart.

Sulphate of magnesia, bicarbonate of potass, and columba. Castor oil.

JAUNDICE.—W. T., æt. 71, Shoemaker. Adm. Sept. 8. Disch. Sept. 23. In H. 15 days. *Recovery.* Dr. Johnson, vol. 19, p. 56. Loss of flesh and appetite 9 months. Lately worried at being out of work. Epigastric pain and soreness 10 days. Jaundice 4 days. Motions pale; no enlargement of liver.

Dilute nitro-muriatic acid and decoction of bark.

JAUNDICE.—E. H., æt. 26, Milliner. Adm. Oct. 14. Disch. Dec. 2. In H. 49 days. *Recovery.* Dr. Garrod, vol. 3, p. 81. In K. C. H. last April with albuminuria. Skin light yellow; slight hepatic tenderness; motions pale; urine 1-8th albumen; tongue coated; venous murmur in neck; albumen persistent.

Sal-volatile, chloric ether and taraxacum. Quinine and bicarbonate of potass. Iodide of potassium and bark.

JAUNDICE.—Emma S., æt. 10. Adm. Sept. 22. Disch. Sept. 30. In H. 8 days. *Recovery.* Dr. Johnson, vol. 17, p. 105. Jaundice and dry cough 4 days. Bowels confined. Motions pale. Pain in lower part of chest. No hepatic tenderness.

Sulphate of magnesia, sp. ammon. arom., and decoction of bark (6 *days*). *Iron and quassia. Aperients.*

JAUNDICE.—T. G., æt. 39, Labourer. Adm. June 8. Disch. July 11. In H. 33 days. *Recovery.* Dr. Beale, vol. 5, p. 192. Had gout twice; cough 3 years. Prev. ill. 3 days. Out of work 2 days before. Skin lemon coloured; motions pale; no enlargement of liver.

Dilute muriatic acid and quinine. Bicarbonate of potass and chloric ether. Aperients. Muriatic acid lotion.

JAUNDICE.—Mary B., æt. 56. Adm. Oct. 30. Disch. Nov. 21. In H. 22 days. *Recovery.* Dr. Beale, vol. 6, p. 328. Subject to sick headache. Prev. ill. 1 week.

Retching. Loss of appetite. Skin deep lemon colour. Considerable itching. No pain, tenderness, or enlargement of liver. Tongue furred.

Dilute muriatic acid. Muriatic lotion applied over liver.

JAUNDICE.—M. A. F., æt. 18, Straw Bonnet Maker. Adm. June 24. Disch. July 4. In H. 10 days. *Recovery.* Dr. Beale, vol. 5, p. 208. Subject to headache. Was frightened 24 days ago and had a fit. Became jaundiced next day. Loss of appetite, constipation, much itching of skin, aching pain over liver for last week. Skin pale lemon colour. Faint basic systolic bruit.

Hydrochloric acid lotion. Pil. col. co. and mist. alba.

JAUNDICE.—W. C., æt. 19, Blacksmith. Adm. Oct. 3. Disch. Oct. 21. In H. 18 days. *Recovery.* Dr. Johnson, vol. 18, p. 19. Drinks spirits freely. Jaundice 12 days. Languor and weakness, nausea and loss of appetite. Skin dark lemon colour; much itching; motions pale; slight pain and tenderness in right hypochondrium; no enlargement of liver.

Podophyllin and colocynth. Dilute nitro-muriatic acid, chloric ether, and bark. Brandy 2 oz.

JAUNDICE.—S. J., æt 21, Waitress. Adm. Sept. 3. Disch. Sept. 11. In H. 8 days. *Recovery.* Dr. Johnson, vol. 19, p. 41. Far advanced in pregnancy. Lately deserted by husband. Jaundice three weeks. Motions at first clay coloured; vomiting; skin deep yellow; much itching; sleeplessness; no hepatic enlargement or tenderness; tongue red. Transferred to lying-in ward on Sept. 11; nearly free from jaundice.

Sulphate of magnesia, dilute sulphuric acid, chloric ether, and sp. meath. pip. Morphia.

JAUNDICE.—A. W., æt. 34, Married. Adm. Oct. 14. Disch. Nov. 4. In H. 21 days. *Recovery.* Dr. Beale, vol. 6, p. 310. Pregnant 4 months. Bowels much confined since. Was "upset" 14 days ago. Soon afterwards headache, loss of appetite, pain round upper part of abdomen, and jaundice. Languid and wakeful. Skin lemon coloured; itching; appetite bad; motions pale: oxalate of lime in urine.

Dilute muriatic acid. Castor oil. Muriatic acid lotion applied over liver.

*JAUNDICE.—W. F., æt. 34, Coachman. Adm. April 27. *Died* on May 5. In H. 8 days. Dr. Johnson, vol. 16, p. 195. Temperate. Vomiting and headache 1 month. Ill 14 days with hepatic pain, languor and jaundice. On 10th day P. 140, and violent delirium, subsiding after stimulants. On adm. slightly jaundiced, edge of liver 2 in. above umbilicus, delirium, subsultus, bowels confined. On 16th day sleepless, petechiæ on trunk and arms, motions pale brown passed involuntarily, tongue dry, brown and fissured. On 21st day dulness, bronchial breathing, and bronchophony over left base, crepitus over right base, increased subsultus, other symptoms as before. *Death* on 22nd day. P.M. Common gall duct the size of a forefinger, its lower end blocked by a gall stone as big as a marble. Liver weighed 4 lbs. 3 oz., ducts distended with pale bile. Interior of gall bladder ulcerated, containing one gall stone. Lower lobes of lungs hepatized, also back part of left upper lobe.

Quinine. Purgatives. Chlorodyne.

*JAUNDICE.—E. H., æt. 33, Married. Adm. May 14. *Died* on June 9. In H. 26 days. Dr. Garrod, vol. 1, p. 104. Jaundice 15 years ago. Became jaundiced 6 days ago, after 3 days pain in right hypochondrium. On adm. deeply jaundiced; liver tender, not enlarged; motions faintly bilious; urine deep yellow; vomiting. Coma on 21st day of jaundice. Death on 24th day. P.M. Liver 2 lb. ¾ oz.; dark red, with large atrophied patches of a gamboge tint. Cystic and bile ducts pervious. Thirteen tetrahedral stones in gall bladder. Two inches of fat on abdomen.

Treatment much varied. Sulphate of magnesia, belladonna, calomel, asafœtida, podophyllin taraxacum.

JAUNDICE—GOUT.—F. M., æt. 44, Publican. Adm. March 9. Disch. April 4.

In H. 26 days. *Recovery.* Dr. Johnson, vol. 15, p. 129. Gouty 8 years; severe general gout 3 years ago. Prev. ill 24 days. Convalescent from an attack of gout, when shivering, vomiting, and jaundice. Motions pale. On adm. jaundiced; slight hepatic pain and tenderness, no enlargement; urine ¼th albumen, containing granular casts and renal epithelium. Free from jaundice about 37th day. An attack of general gout on 38th day. Free from pain on 47th day.

Liq. ammon. acetat. and citrate of iron (39 *days*). *Then bicarbonate of potass and carbonate of ammonia. Colchicum.*

JAUNDICE.—E. L., æt. 28, Servant. Adm. March 3. Disch. April 4. In H. 32 days. *Recovery.* Dr. Beale, vol. 5, p. 134. Languid and out of health lately. Ill 8 days with vomiting, headache, pain in liver and right shoulder, and jaundice. On adm. deeply jaundiced; motions white. Liver enlarged and tender. Gall bladder much distended about 15th day. Recovered perfectly.

Dilute muriatic acid. Mist. alba.

HYDATID TUMOUR OF LIVER.—C. C., æt. 18, Costermonger. Adm. July 13. Disch. Aug. 8. In H. 26 days. *Unrelieved.* Dr. Garrod, vol. 1, p. 171. Globular, smooth, elastic tumour in right hypochondrium, and a larger tumour lower down on same side. Received a blow with fist in right flank 1 wk. before their appearance, 8 months ago. Systolic bruit at heart's apex. (Rheum. 4 yrs. ago.) General health good.

Syrup of iodide of iron. Biniodide of mercury ointment. Belladonna plaster.

HYDATID TUMOUR OF LIVER.—S. M., æt. 24, Tailoress. Adm. June 18. Disch. July 1. In H. 13 days. *Unrelieved.* Dr. Beale, vol. 5, p. 206. Received a kick in right side 9 years ago. Pain and swelling of side 12 months afterwards. Tapped twice, 5 years ago; 4½ pints fluid drawn off altogether. Hepatic dulness level with right nipple. Right side at level of ensiform cartilage, 1 in. larger than left. Discharged at her own request.

Sesquichloride of iron.

CANCER OF LIVER.—J. F., æt. 30, Servant. Adm. May 11. Disch. June 17. In H. 57 days. *Unrelieved.* Dr. Johnson, vol. 17, p. 25. Gout 7 or 8 times during last 20 years. Cough and loss of flesh 3½ months. Nausea and vomiting 2½ months. Hepatic enlargement 1 month. On adm. edge of liver felt 2 in. above umbilicus; hepatic dulness to just below right nipple. No jaundice or ascites. Urine albuminous.

Haust. effervescens (11 *days*). *Sulphate of magnesia and sulphate of iron. Compound iodine ointment over liver.*

CANCER OF LIVER.—J. H., æt. 57, Wood Carver. Adm. May 9. *Died* on May 11. In H. 2 days. Dr. Johnson, vol. 17, p. 13. Intemperate. Ill 2 months, with hepatic pain, followed by ascites and jaundice. Œdema of legs, 1 week. Sleepless for 3 nights before adm. On adm. deeply jaundiced. Edge of liver extending nearly to umbilicus. Diarrhœa, dry tongue, and P. 130 on day after adm. P.M.—Considerable ascites. Liver, 12 lbs. 6 oz., infiltrated with cancer. Mesenteric glands cancerous. Heart large, 18 oz. Aorta bony and atheromatous.

SCROFULOUS ENLARGEMENT OF LIVER.—T. G., æt. 18, Foundryman. Adm. June 18. Disch. Sept. 12. In H. 86 days. *Unrelieved.* Dr. Johnson, vol. 17, p. 65. Enlarged abdomen 12 years. Severe epistaxis 9 months ago. Cough 6 months. Very pallid. Skin dry. Edge of liver 1 in. below umbilicus. Moveable tumour on left side. Urine ½ albumen, containing granular casts. Flatulence, waterbrash, and constipation. Frequent epistaxis.

Sulphate of iron, quinine, and dilute sulphuric acid. Turpentine. Gallic acid snuff.

Enlarged Liver.—A. M., æt. 48, Nurse. Adm. Aug. 6. Disch. Sept. 12. In H. 37 days. *Unrelieved.* Dr. Johnson. Prev. ill. 12 months. Loss of flesh and strength, cough and expectoration, and occasionally pain in abdomen, and very slight jaundice. Liver much enlarged. Lower edge rough. Dulness, harsh breathing, and rhoncus under left clavicle. P. 76.

Gentian and bicarbonate of soda. Aromatic spts. of ammonia, carbonate of soda, and peppermint. Colocynth and Taraxacum.

Cirrhosis.—J. C., æt. 52, Wheelwright. Adm. July 28. *Died* Aug. 31. In H. 34 days. Dr. Johnson, vol. 19, p. 16. Has drunk freely for last 7 years. Winter cough 6 years. Œdema of legs and enlarged abdomen 2 months. Jaundice 6 weeks. Itching of skin. Liver enlarged. Motions pale and relaxed. The jaundice became deeper, the dropsy rapidly increased; he became very weak, and died suddenly. Urine free from albumen throughout. P.M.—Much fluid in abdomen. Lungs very emphysematous. Liver, 3 lbs. 6 oz., pale, firm, and much cirrhosed. Kidneys large, healthy.

Quinine, hydrochloric acid, and chloric ether (15 *days*). *Acetate of potass, squills, juniper, broom tops, and digitalis. Compound jalap powder. Gin,* 6 *oz. Legs punctured.*

Cirrhosis of Liver.—E. S., æt. 37, Flower Seller. Adm. April 22. Disch. May 9. In H. 17 days. *Relieved.* Dr. Johnson, vol. 15, p. 173. Excessive spirit drinking. Ill 2 months with jaundice, hepatic pain, and abdominal enlargement. On adm. skin dry. Slight jaundice. Ascites. Superficial abdominal veins enlarged. Hepatic dulness diminished.

Squills, digitalis, juniper, and broom tops.

Ascites.—G. D., æt. 29, Stoker. Adm. June 17. *Died* Aug. 3. In H. 47 days. Dr. Garrod, vol. 1, p. 142. Dropsy of belly and legs 1 year. Tapped 8 months, and again 3 weeks ago. Pallid. Has angular and lateral curvature of spine. Abdomen 43 inches in circumference. Skin dry. Crepitation at apices of lungs. Heart's apex to left of nipple. No bruit. Urine not albuminous. Tapped on July 2, and again on July 28. Sank soon after 2nd operation.

Syrup of iodide of iron. Carbonate of ammonia and chloric ether. Bitartrate of potass and jalap.

Perforation of Cœcum.—J. Y., æt. 44, Shoemaker. Adm. Dec. 23. *Died* on Dec. 23. Dr. Johnson, vol. 18, p. 150. No alvine evacuation for 20 days. Vomiting and colicky pains 14 days. Sudden severe pain in abdomen about 6 a.m., on morning of adm. In a state of collapse when adm.; some vomiting, abdomen acutely painful and tender. Died at 2.30 p.m. P.M.—Recent general peritonitis. Colon enormously distended by fæces, some of which had escaped through a small aperture in cœcum. Intestine around perforation of dark claret colour. Stricture of rectum 8 inches above anus.

Opium. Brandy.

Peritonitis.—B. D., æt. 43, Laundress. Adm. Oct. 17. Disch. Oct. 31. In H. 14 days. *Recovery.* Dr. Johnson, vol. 19, p. 121. General pain and tenderness of abdomen, after a kick in left groin, 6 days ago. P. 110, small. Bowels open. No vomiting.

Opiate poultices. Quinine and opium. Ammonia and chloric ether. Brandy. Afterwards bark and carbonate of ammonia.

Constipation.—Peritonitis.—W. H., æt. 22, Potman. Adm. May 21. Disch. July 1. In H. 41 days. *Recovery.* Dr. Beale, vol. 6, p. 168. Prev. ill. 3 days. Constipation, great pain and tenderness of abdomen, and tympanites.

Castor oil and warm water enema. Morphia. Calomel. Afterwards bicarbonate of soda and hydrocyanic acid.

Constipation.—C. W., æt. 22, Porter. Adm. July 31. Disch. Aug. 8. In H. 8 days. *Recovery.* Dr. Johnson, vol. 17, p. 83. Prev. ill. 5 days. Cœcal pain and tenderness, constipation, and vomiting.
Castor oil and warm water enema. Morphia.

Constipation.—Wm. P., æt. 22. Adm. Sept. 11. Disch. Sept. 16. In H. 5 days. *Recovery.* Dr. Johnson. Frequent constipation 4 months. Pain and tenderness of cœcum. Bowels confined 3 days.
Castor oil and warm water enema.

Colic?—E. W., æt. 52, Ticket Writer. Adm. Nov. 15. Disch. Nov 18. In H. 3 days. *Recovery.* Dr. Johnson, vol, 17, p. 95. Pain and tenderness over liver 3 months. Vomiting at times. Nausea 1 week. Much abdominal pain on morning of adm. Liver enlarged. Stools clay-coloured. Twenty years ago was jaundiced, and passed gall-stones.
Warm bath. Castor oil and laudanum. Turpentine stupes. Soda and hydrocyanic acids.

Colic.—Joseph N., æt. 28. Adm. Oct. 23. Disch. Oct. 28. In H. 5 days *Recovery.* Dr. Johnson.
Dilute nitro-muriatic acid, chloric ether, and bark.

Diarrhœa.—Anne F., æt. 42. Adm. Dec. 7. Disch. 16. In H. 9 days. *Recovery.* Dr. Beale, vol. 7, p. 22. Diarrhœa 14 days ago. Now ill 2 days. Headache. Purging till 3rd day after adm. Tongue white. P. 92. Crepitation at bases of lungs.
Liquor ammoniæ acetatis and chloric ether. Then sesquichloride of iron and quassia.

Diarrhœa.—Sarah W., æt. 23. Adm. July 18. Disch. Aug. 12. In H. 25 days. *Recovery.* Dr. Garrod, vol. 1, p. 183. Epileptic. Diarrhœa 4 days; well on 4th day after adm. Headache.
Asafœtida.

Diarrhœa.—Charles S., æt. 52, Teacher of Music. Adm. Sept. 26. Disch. Sept. 30. In H. 4 days. *Recovery.* Dr. Beale, vol. 5, p. 280. Diarrhœa 4 days; well on 3rd day after adm.
Opium and logwood.

Dysentery.—T. T., æt. 29, Fisherman. Adm. October 28. Disch. Nov. 2. In H. 5 days. *Much relieved.* Dr. Johnson, vol. 17, p. 137. Had ague 5 months ago. Prev. ill. 3 months. Blood and mucus in motions; frequent griping and diarrhœa. Tongue furred. Urine loaded with lithates.
Opium.

Dysentery.—T. C., æt. 32, Steamboat Steward. Adm. June 2. Disch. July 1. In H. 29 days. *Relieved.* Dr. Johnson, vol. 16, p. 237. Had acute rheum. 3 times. Slight dysentery in India 4 years ago. Prev. ill. 3 months. Pain and tenesmus. Blood and mucus in motions.
Logwood. Sesquichloride of iron and chlorate of potass. Conf. pip. nig.

Renal Abscess.—Sarah A., æt. 22, Cook. Adm. Feb. 16. Disch. Aug. 22. In H. 187 days. *Relieved.* Dr. Beale, vol. 6, p. 90. Pain in right loin for 2½ years, extending down right thigh and leg during last 3 months. Two or three rigors daily of late. Flushed. Fulness and tenderness in right lumbar region. Micturition painful and frequent (varying from every hour to every ¼ hour), followed by vesical pain. Urine containing albumen and pus (varying from ʒvj to

℥ij daily). Catamenia suppressed after first week in hosp. Appetite good. Sleep disturbed.

Sesquichloride of iron. Bicarbonate of potass. Dilute muriatic acid. Sedatives. Aperients. Warm hip baths.

Renal Calculus.—G. M., æt. 18, Wood Cutter. Adm. May 8. Disch. May 30. In H. 22 days. *Relieved.* Dr. Garrod, vol. 1, p. 102. Paroxysms of pain in left loin, with retraction of testicle, 12 years. Occasional hœmaturia 2 years. Attacks brought on by jolting exercise. On adm. urine albuminous and bloody (for 2 days after). Severe nephralgia on 4th day.

Purgatives. Sinapisms to loin. Seton in left loin on May 12.

Lithic Acid Gravel.—E. B., æt. 44, Groom. Adm. Sept. 17. Disch. Nov. 11. In H. 55 days. *Very much relieved.* Dr. Beale, vol. 6, p. 260. Has drunk hard. Subject to nephralgic attacks 10 years. In K. C. H. 18 months ago. Much pain and tenderness of abdomen, esp. about liver. Urine pale; no albumen. Copious and persistent sediment of uric acid 7 days later. Occasional severe nephralgia, with retraction of testicle.

Alkalies (19 *days*). *Dilute hydrochloric acid* (16 *days*). *Benzoic acid. Opiates. Purgatives. Warm baths and fomentations.*

Hœmaturia.—William C., æt. 8. Adm. April 13. Disch. May 5. In H. 22 days. *Recovery.* Dr. Johnson, vol. 15, p. 165. Bruise on left loin 3 years ago, followed by nausea and vomiting. Occasional hœmaturia, especially after exertion. Blood casts and globules, and a trace of albumen in urine for a few days. Lithic acid sediment on May 1.

Renal Disease.—Pleurisy.—E. S., æt. 29, Waiter. Adm. April 16. *Died* on May 7. In H. 21 days. Dr. Johnson, vol. 15, p. 167. Intemperate. Three years ago dropsy for 8 months, after acute rheumatism, returning 3 months after recovery. Now pain in right side and dyspnœa. Dulness over right lung, below level of scapular angle, breathing there feeble and vesicular at first, then bronchial; pleuritic rub behind. Headache and nausea. Urine containing granular casts, and ⅔ albumen. *Died* on 21st day. P.M.—Fluid in right pleura. Right lung gorged and recently adherent. Commencing atrophy of kidneys.

Squills and sesquichloride of iron. Then strychnia and sesquichloride of iron. Brandy. Hot air baths.

Acute Renal Dropsy.—M. B., æt. 55, Charwoman. Adm. April 21. Disch. June 30. *Relieved.* Dr. Johnson, vol. 15, p. 171. 6 weeks ago pain in loins, vomiting, headache, and dropsy. On adm. urine containing blood, oil, epithelial, waxy and granular casts. Almost free from dropsy on 22nd day. Gout about right ankle on 35th day (10 days), and in right great toe on 55th day.

Liq. ammon. acetatis (6 *days*). *Then chlorate of potass and sesquichloride of iron. Hot air baths.*

Acute Renal Dropsy.—J. F., æt. 36, Hospital Nurse. Adm. March 5. Disch. May 11. In H. 67 days. *Much relieved.* Dr. Beale, vol. 6, p. 115. Headache and sore throat 3 weeks. Urine brown and scanty (10 days). Had been with a case of scarlet fever. Slight anasarca. Blood casts and much albumen in urine. On 30th day much blood, small waxy casts, renal epithelium, and oil. Urine almost normal on 46th day.

Quinine and sesquichloride of iron. Cod-liver oil.

Acute Renal Dropsy.—J. C., æt. 27, Needlewoman. Adm. March 16. Disch. April 1. In H. 16 days. *Recovery.* Dr. Garrod, vol. 1, p. 66. Caught cold 8 days ago. Then vomiting, headache, and œdema of legs. Urine containing small waxy casts, renal epithelium, and a trace of albumen.

Liq. ammoniæ acetatis (9 *days*). *Then same with citrate of iron. Jalap and bitartrate of potass powders.*

Acute Renal Dropsy.—Mary K., æt. 47, Married. Adm. Jan. 28. Disch. April 4. In H. 66 days. *Recovery.* Dr. Beale, vol. 6, p. 78. Prev. ill. 5 days. Pain in loins. Œdema of legs, followed by erysipelas about left ankle. P. 96. Urine containing albumen (till 38th day) blood. Epithelial casts and lithic acid.

Liquor ammoniæ acetatis and chloric ether. Afterwards sesquichloride of iron. Compound jalap powder.

Acute Renal Dropsy.—R. C., æt. 59. Adm. Feb. 9. Disch. April 4. In H. 44 days. *Recovery.* Dr. Garrod, vol. 2, p. 50. Attacks of dropsy 9, 3, and 2 years ago. Now anasarcous and ascitic 3 weeks. Pallor, basic systolic bruit over heart, drowsiness and headache. Urine very albuminous (till 42nd day) and containing renal epithelium, small waxy casts, and oil.

Bitartrate of potass and jalap powder. Ammoniated tincture of guaiacum. Exposed to cold after discharge. Fresh dropsy and albuminuria. Re-admitted on April 7. Disch. *well* on April 25.

Acetate and carbonate of ammonia (7 *days*). *Then ammoniated tincture of guaiacum.*

Acute Renal Dropsy.—A. C., æt. 20, Laundress. Adm. Feb. 25. Disch. April 11. In H. 45 days. *Recovery.* Dr. Garrod, vol. 2, p. 56. Dropsy 3 years ago. Prev. ill. 6 days. Got wet. Then shivering, headache, nausea, slight anasarca. Urine semi-solid on boiling, contained blood globules and casts. Systolic bruit at apex of heart.

Sesquichloride of iron. Sulphate of iron. Ammoniated tincture of guaiacum. Bitartrate of potash and jalap powders. Sinapisms to loins. Hot air baths.

Acute Renal Dropsy.—E. J., æt. 26, Cook. Adm. June 20. Disch. Sept. 16. In H. 88 days. *Recovery.* Dr. Beale, vol. 6, p. 200. Epigastric pain, worse after food, and pain in loins, 1 month. Vomited 2 oz. dark blood 14 days ago. Slight œdema of legs and face, 8 days. Very pallid. Urine smoky, ½ albumen. Sour risings and flatulence. Appetite good.

Liq. ammon. acetatis. Sesquichloride of iron. Hot air baths. Dry cupping. Aperients.

Acute Renal Dropsy.—C. F., æt. 50, Charwoman. Adm. May 16. Disch. Aug. 8. In H. 84 days. *Relieved.* Dr. Beale, vol. 5, p. 166. Typhus 25 years ago. Dropsy (2 months) 2 years ago. Prev. ill. 7 days. Loss of appetite. Urine scanty and albuminous, cont. blood and granular casts. Free from dropsy 3 weeks before disch., but albumen did not disappear.

Liq. ammon. acetat. (6 *days*). *Then same with sesquichloride of iron. Purgatives. Hot air baths. Loins dry cupped.*

Acute Renal Dropsy.—J. D., æt. 41, Whitesmith. Adm. Oct. 21. Disch. Oct. 29. In H. 8 days. *Recovery.* Dr. Johnson, vol. 18, p. 55. Had scarlatina and typhus. Cephalalgia 6 months. Prev. ill. 3 days. Commenced in face. Urine scanty at first. On adm. anasarca and slight ascites. Physical signs of emphysema. Crep. at bases of lungs. Urine slightly albuminous cont. granular epithelial and waxy casts, much renal epithelium, and a little blood.

Compound jalap powder. Hot air baths.

Acute Renal Dropsy.—H. S., æt. 68, Labourer. Adm. Oct. 1. Disch. Oct. 24. In H. 23 days. *Recovery.* Dr. Johnson, vol. 19, p. 100. Subject to pain in loins some years. Exposed to wet and cold. Prev. ill. 3 weeks. Urine at first dark brown. Legs very œdematous on adm. Pain in left side. Some dulness and feeble breathing at bases. Urine pale, ⅓rd. albumen, cont. oily casts 7 days later. P. 52. Appetite bad. Well 5 days before disch.

Compound jalap powder. Liq. ammon. acetatis (*afterwards with T. Ferri Mur.*) *Hot air baths.*

Acute Renal Dropsy.—H. R., æt. 44. Printer. Adm. Oct. 24. *Died* on Nov. 12. In H. 19 days. Dr. Johnson, vol. 19, p. 127. Dropsy after scarlatina, when 40 years of age. Rather intemperate. Prev. ill. 12 days. Tightness at chest. Much dyspnœa. Anæmia. General dropsy, great œdema of legs. Urine scanty and smoky, cont. $\frac{1}{4}$th albumen, waxy and granular casts, blood, pus, and renal epithelium. Some oil 16 days later. Dulness, slight crep., and feeble breathing at right posterior base. 13 days later, increased dulness and crep. over right base, some dulness and coarse crep. over left base. Death from increase of dyspnœa.

Sesquichloride of iron with digitalis quinine and muriate of ammonia in turn. Then Vin. A. P. T. and liq. ammon. acetatis. Compound jalap powder. Loins dry cupped. Sinapisms. Hot air baths.

Acute Renal Dropsy.—J. M., æt. 42. Barman. Adm. Nov. 19. Disch. Dec. 23. In H. 34 days. *Recovery.* Dr. Johnson, vol. 18, p. 107. Since second attack of acute rheumatism, 6 years ago, has been subject to vomiting, lumbar pain, palpitation, and œdema of legs. On adm. urine smoky, albumen $\frac{1}{8}$. Pain in loins. Legs slightly dropsical. P. 92. Basic systolic bruit. Has emphysema. 29 days later, urine free from blood, albumen $\frac{1}{16}$, no dropsy.

Liquor ammon. acetatis, alone, and with citrate of iron. Sesquichloride of iron. Tannic acid. Pulv. Jalapæ Co. Hot air baths.

Acute Renal Dropsy.—G. M., æt. 41. Lawyer's Clerk. Adm. Nov. 7. Disch. Dec. 5. In H. 28 days. *Recovery.* Dr. Beale, vol. 5, p. 345. Prev. ill. 4 weeks. Shivering, headache, anorexia, dropsy, scanty and high coloured urine. Now considerable œdema of legs. Urine smoky, $\frac{1}{8}$ albumen, cont. blood, granular casts, and uric acid.

Sesquichloride of iron. Jalap of scammony powder. Hot air baths. Loins dry cupped.

Acute Renal Dropsy.—A. D., æt. 23. Cook, Adm. June 22. Disch. July 22. In H. 30 days. *Recovery.* Dr. Johnson, vol. 17, p, 69. Prev. ill. 7 days. Thirst, anorexia, vomiting, headache, considerable œdema of legs, face puffy. Urine pale, $\frac{1}{2}$ albumen, cont. pale granular casts on adm., blood, and blood casts 11 days later. Free from dropsy on the 14th day after adm.

Chloric ether and liq. ammon. acetatis (10 days), Then sesquichloride of iron. Hot air baths. Loins dry cupped. Pil. Col. Co.

Chronic Renal Disease.—W. T., æt. 34. Porter. Adm. Nov. 6. Disch. Nov. 28. In H. 23 days. *Relieved.* Dr. Johnson, vol. 18, p. 83. In K.C.H. with renal dropsy 21 months ago. Erysipelas (face and right leg) 3 months ago. Lumbar pain and œdema of legs 3 months. Urine at times high coloured, now plentiful, $\frac{1}{3}$ albumen, sediment of medium and large granular casts, renal epith., and pus. crep. at bases of lungs, cough and expectoration on disch. No dropsy, alb. $\frac{1}{2}$.

Quinine, sesquichloride of iron, and muriate of ammonia. Compound jalap powder. Hot air baths.

Renal Dropsy.—T. P., æt. 26, Light Porter. Adm. April 4. *Died* on April 15. In H. 11 days. Dr. Beale. Œdema of legs 3 weeks. Nocturnal micturition 1 week. Very pallid and anasarcous. Dry cough. Urine pale and albuminous ($\frac{1}{2}$) cont. oily casts and cells. On 9th day, severe abdominal pain, vomiting, thready pulse, and anxious countenance, rapid sinking, death on 11th day. No P. M.

Carbonate of ammonia and chloric ether. Sinapisms to abdomen.

Renal Dropsy.—Jane R., æt. 34. Cook. Adm. Feb. 18. *Died* on April 4. In H. 45 days. Dr. Johnson, vol. 15, p. 105. Ascites and œdema of legs 3 months, following a cold. Very pallid. Emphysematous. Systolic bruit loudest at apex of heart. No rheum. Urine $\frac{1}{4}$ albumen. On 7th day free from dropsy, albumen $\frac{2}{3}$. After 16th day, vomiting, purging, headache, drowsiness, at first intermittent,

then constant. From 22nd day, wine about 10oz. daily. Melœna on 40th day. Abdominal pain and tenderness on 42nd day. Death on 45th day.

Iron and ammonia. Iodide of potassium and ammonio-citrate of iron. Hydrocyanic acid, ice, and creosote to check vomiting.

Renal Dropsy.—M. A. S., æt. 25. Schoolmistress. Adm. Nov. 18. Disch. Nov. 28. In H. 10 days. *Much relieved.* Dr. Johnson, vol. 18, p. 103. Prev. ill. 2 months. Nausea, vomiting, pyrosis, and flatulence. On adm., epigastrium tender, bowels confined, urine very albuminous. Œdema of ankles 5 days later. No vomiting after 2nd day. Albumen decreased.

Dilute hydrocyanic acid. Muriate of ammonia and citrate of iron. Pil. Cal. c. Col. Sinapism to epigastrium.

Renal Dropsy.—E. B., æt. 56. Laundress. Adm. Oct. 5. Disch. Oct. 31. In H. 26 days. *Recovery.* Dr. Johnson, vol. 19, p. 106. Ankles œdematous at times for some years past. Occasional vomiting. Urine scanty 2 months, never high coloured. Legs œdematous 14 days. On adm. urine pale, of sp. gr. 1026, ¼ albumen, sediment of small waxy and epithelial casts. 9 days later, less dropsy, urine free from albumen.

Chloric ether, sal volatile, and citrate of iron. Then quinine and sesquichloride of iron. Hot air baths.

Renal Dropsy (Chronic).—Ann F., æt. 37. Bootbinder. Adm. July 11. Disch. Aug. 15. In H. 35 days. *Unrelieved.* Dr. Johnson, vol. 15, p. 274. Acute dropsy from cold 2 years ago. Second attack 1 year ago. Urine thin, dark coloured. Legs œdematous since. On adm. urine ½ albumen, copious deposit of large granular and waxy casts, oil cells, and pus. Anasarca, pain in loins, constipation, bad appetite.

Sesquichloride of iron. Gallic acid. Purgatives.

Readm. Oct. 7. *Died* Oct. 18. In H. 11 days. Dr. Johnson, vol. 19, p. 104. Scarcely any dropsy now. Has been subject to headache and vomiting since disch. Urine ⅓ albumen, sediment as before. Much thirst and vomiting. Heart's impulse strong. Quantity of urine decreased. Death from exhaustion. P.M.—Bases of lungs œdematous. Old tubercles in apex. Left ventricle much hypertrophied. An old hydatid cyst in back of right lobe of liver. Kidneys granular, wasted, "waxy."

Liq. ammon. acetat. and sulphate of magnesia. Sulphuric acid, and sulphate of magnesia. Dilute nitro-muriatic acid. Calomel and jalap. Brandy. Loins dry cupped.

Renal Dropsy.—Emma F., æt. 26, married. Adm. May 23. *Died* Aug. 14. In H. 83 days. Dr. Johnson, vol. 16, p. 217. Had scarlet fever when a child. Out of health 4 years. Œdema of ankles 2¼ years. Epileptic fits 8 months ago (2 while in hosp.) Nightly startings 4 months. Diarrhœa 14 days. Constant vomiting. Urine of sp. gr. 1010, ammoniacal, cont. a little blood and albumen. *After 74th day severe diarrhœa,* increasing drowsiness, coma, and death.

Dilute hydrochloric acid with iron, quinine, and buchu and henbane in turn. Benzoate of ammonia. Dilute phosphoric acid and phosphate of iron. Krameria and logwood.

Renal Dropsy.—J. S., æt. 34. Waiter. Adm. June 4. *Died* on July 17. In H. 43 days. Dr. Beale, vol. 5, p. 186. Had gout twice. Prev. ill. 2½ months. Headache. Constant vomiting. Considerable œdema of legs, face puffy, slight ascites. Urine pale and plentiful, sp. gr. 1008, cont. ¼ albumen, and pale waxy casts. On 9th day after adm. gout in left great toe. 30 days later loud to and fro sound over heart, cardiac dulness increased. 2 days later, 2 epileptic fits—next day dulness and br. br. over lower half of left lung. Death from apnœa. *P.M.*—Pericardium contained 1 pint fluid. Thick recent lymph on heart and sac. Weight of heart 19 oz., left vent. hypertrophied. Much fluid in left pleura—lower ¾ of lung carnefied. Kidneys granular, wasted, and fatty.

Sesquichloride of iron. Liq. ammon. acetatis and carbonate of ammonia. Creosote and muriate of morphia. Dilute hydrocyanic acid and Haust. efferv. Brandy to 12 *oz.*

Diabetes Mellitus.—F. S., æt. 32. Chimney Sweep. Adm. July 16. Disch. July 28. In H. 12 days. *Relieved.* Dr. Garrod, vol. 1, p. 177. Very intemperate. Prev. ill. 6 months. Thirst very troublesome, loss of strength, ravenous appetite, skin dry, legs slightly œdematous, ulcers inside lips, bowels relaxed. Passing 13 pts. urine in 24 hours, sp. gr. 1035, extremely saccharine. Slight dulness, harsh breathing, and prolonged expiration under right clavicle. Quantity of urine fell to 32 oz.

Non-saccharine diet. Dilute phosphoric acid.

Diabetes Mellitus.—E. G., æt. 19. Silk Sewer. Adm. Oct. 23. Disch. Dec. 2. In H. 40 days. *Relieved.* Dr. Johnson, vol. 19, p. 123. Ill 6 weeks. Thirst, emaciation, and copious urine. Now rather thin, very thirsty, appetite good, skin dry, bowels and cata. regular. Passes 8 pts. urine daily, sp. gr. 1035, very saccharine. Gums spongy. Slight cough and morning expectoration. Dulness, prolonged expiration, increased vocal resonance, and slight click at right apex. Urine decreased. On Nov. 26, passed 32 oz. sp. gr. 1028.

Quinine and sesquichloride of iron Cod-liver oil. Non-saccharine diet.

Diabetes Mellitus.—A. G., æt. 52, Picture Frame Maker. Adm. July 7. *Died* Aug. 21. In H. 45 days. Dr. Beale, vol. 6, p. 222. Always epileptic. Loss of strength 2 years. Frequent micturition and intense thirst 6 months. $2\frac{1}{2}$ months ago was passing 5 pts. urine daily, and had cough and expectoration, with crepitation under right clavicle. Now has intense thirst; good appetite; tongue furred and dry; skin dry; bowels much confined; edge of liver felt at umbilicus. No cough; slight crep. under clavicle sometimes. Urine $8\frac{3}{4}$ pts. daily; sp. gr., 1036-1045, very saccharine. Apthæ formed on tongue and fauces. A crop of boils appeared about Aug. 16. A large boil on right wrist was followed by phlegmonous erysipelas of right arm, exhaustion and death. P.M.—Old adhesions of pleuræ. Scanty tubercle at right apex. Pericardium everywhere firmly adherent. Liver large, 7lbs. 2 oz., chocolate coloured, and firm; capsule opaque. Kidneys congested. Right supra renal capsule very large ($\frac{1}{4}$ *oz*). Excess of subarachnoid fluid. Basilar and postr. cerebral arteries very atheromatous. Cysts and chalky bodies in choroid plexuses.

Non-saccharine diet.

Typhus.—Mary A., æt. 18, Servant. Adm. Dec. 5. *Died* on Dec. 18. In H. 13 days. Dr. Johnson, vol. 18, p. 123, Prev. ill. 4 days. Shivering and prostration of strength, Sleepless and delirious on 3rd and 4th nights On 5th day P. 108, R. 24; rubeoloid eruption on trunk well marked on 7th day; tongue yellowish brown. On 10th, rhonchus and sibilus all over chest. P. 112, R. 40, will not protrude tongue. On 11th unconscious, subsultus, numerous petechiæ, tongue dry, brown, and cracked. On 16th day only slight delirium. Two small sores on back. P. 96, R. 24; lungs clear. Afterwards parotid swelling and abscess. Conscious till shortly before death on 18th day.

Dilute nitro-muriatic acid and bark. Quinine enemata. Brandy to 12 *oz. Morphia. Back dry cupped. Abscess opened.*

Typhus.—H. L., æt. 25, Printer. Adm. June 29. *Died* on July 13. In H. 14 days. Dr. Garrod, vol. 1. p. 157. Prev. ill. 3 days. Shivering, loss of appetite, vertigo, sleeplessness. Diarrhœa on 3rd day. On 5th day very weak. Skin hot; thick, white fur on tongue; urine albuminous. Indistinct rubeoloid rash on 6th day. On 9th day crepitus and slight dulness at right base. On 10th day, P. 112, R. 56. On 11th day crepitation, and on the 13th br. br. over both bases, up to angle of scapula. On 14th day three loose motions. P. 140, R. 92. Death on 17th day. P.M.—Right lower pulmonary lobe hepatized, and left nearly so. Upper lobes gorged. A little fluid in pleuræ and pericardium.

Mist. camph. (3 *days*). *Quinine and sulphuric acid* (5 *days*). *Carbonate of ammonia, chloric ether, and senega. Brandy up to* 24 *oz.*

Typhus.—S. H., æt. 22, Student. Adm. June 28. *Died* on July 3. In H. 5 days. Dr. Johnson. Prev. ill. about 8 days. Delirious on first night. Severe headache. Slight measly rash on abdomen on 7th day. Very marked on 10th day. On 9th day very delirious. P. 104, feeble. Violent delirium on 9th and 10th nights. On 11th day, P. 104, R. 36. On 13th day tympanites, subsultus, unconsciousness. Death on 14th day.

Dilute nitro-muriatic acid, liq. cinchonæ, and chloric ether. Brandy, 18 *oz. on adm., afterwards increased to* 24 *oz. Morphia. Turpentine stupes to abdomen. Rue enema.*

Typhus.—Thomas C., æt. 43. Adm. Aug. 24. Disch. Sept. 23. In H. 30 days. *Recovery.* Dr. Johnson. Wife and daughter in K. C. H. with typhus. Extremely deaf. Highest pulse 120. Highest resp. 34.

Dilute nitro-muriatic acid, liquor cinchonæ, and chloric ether. Afterwards quinine and acid. Opiate enema. Brandy to 9 *oz. Turpentine stupes.*

Typhus.—Rachel C., æt. 34, Married. Adm. July 30. Disch. Sept. 19. In H. 51 days. *Recovery.* Dr. Johnson, vol. 17, p. 59. Access gradual during last 3 weeks. Delirious at times, 1 week. Had support. Hot and flushed. Copious rubeoloid and petechial eruption. Tongue dry and brown. P. 108. Free from delirium after night of 30th, and on Aug. 5. P. 80. Tongue nearly clean.

Dilute nitro-muriatic acid, liquor cinchonæ, and chloric ether (18 *days*). *Then quinine. Brandy,* 8 *oz.*

Typhus.—Rachel C., æt. 15. Adm. Aug. 17. Disch. Oct. 3. In H. 47 days. *Recovery.* Dr. Johnson. Mother now in hosp. with typhus. Prev. ill. 6 days, Epileptoid convulsions on 1st and 2nd days. Cough, sore throat, prostration, loss of appetite. On adm. thick rubeoloid eruption on trunk and limbs. Tongue dry and brown. Throat sore. P. 124, R. 60. Rhonchus and crepitus over back of lungs. Trace of albumen in urine.

Dilute nitro-muriatic acid, liquor cinchonæ, and chloric ether (3 *days*). *Same with turpentine* (24 *days*). *Afterwards quinine. Opiate enema. Brandy to* 12 *oz.*

Typhus.—M. D., æt. 64, Shoemaker. Adm. Nov. 4. *Died* on Nov. 8. In H. 4 days. Dr. Beale, vol. 5, p. 343. Prev. ill. 3 days. On 1st day shivering, great prostration of strength, and unconsciousness for some hours. Semi-conscious on adm. Tongue dry and brown. P. 96, feeble. Indistinct mottling of skin. Urine not albuminous, passed under him. Increasing coma. Death on 7th day. P.M.—Lower lobes of lungs congested. Spleen friable. Liver soft, other organs healthy.

Liq. ammon. acetatis, sp. ammon. arom., and chloric ether. Brandy 12 *oz.*

Typhus.—J. B., æt. 54. Adm. Sept. 16. *Died* on Sept. 19. In H. 3 days. Dr. Beale, vol. 6, p. 272. Ill fed lately. Two fever cases in same house. Prev. ill. 1 week. Shivering. Severe diarrhœa on 4th and 5th days. Tongue dry and coated. Considerable abdominal tenderness. Slight tympanites. Indistinct rubeoloid eruption. P. feeble and irregular on 9th day. Death on 10th day. P.M.—Lower pulmonary lobes congested. Spleen enlarged, dark, and friable.

Chlorate of potass, dilute nitro-muriatic acid, and liquor cinchonæ. Opiate nemata. Brandy to 6 *oz.*

Typhus.—Elizabeth H., æt. 30. Adm. Oct. 20. *Died* on Oct. 21. Dr. Johnson. Almost moribund on adm. P. 152, R. 48.

Dilute nitro-muriatic acid, chloric ether, and decoction of bark. Brandy, 12 *oz.*

Typhus.—J. H., Student. Adm. Oct. 5. Disch. Nov. 7. In H. 33 days. *Recovery.* Dr. Johnson. Exposed to contagion but 14 days ago. Prev. ill. 4 days. Eruption appeared on Oct. 3. Highest pulse, 100. Highest resp., 38.

Liquor cinchonæ, dilute muriatic acid, and chloric ether (5 *days*). *Same with quinine* (19 *days*). *Then quinine and iron. Brandy to* 9 *oz. Aperients.*

Typhus.—John D., æt. 13. Adm. June 3. Disch. July 8. In H. 35 days. *Recovery.* Dr. Johnson, vol. 17, p. 49. Ill fed lately. Prev. ill 10 days. Headache and vomiting, sore throat on 6th day. Faint rubeoloid rash. Tongue brown. Urine albuminous on 11th day (7 days). On 12th day, P. 132, R. 40. Delirious 16th night. Convalescent 30th day.

Dilute nitro-muriatic acid and chloric ether, afterwards with quinine and nitre (13 *days*). *Then iron. Brandy* 6 *oz.*

Typhus.—W. H., æt. 30, Wood Cutter. Adm. Oct. 13. Disch. Nov. 25. In H. 43 days. *Recovery.* Dr. Johnson, vol. 18, p. 33. Prev. ill. 4 days. Commenced with shivering. Rubeoloid eruption well marked, appeared on 5th day. Delirium on 3rd night, and on 7th till 14th day. Rhonchus and sibilus over chest; much deafness. P. 120 on 7th day. Trace of albumen in urine on 11th day. Slow improvement after 15th day.

Dilute nitro-muriatic acid, chloric ether, and liquor cinchonæ (20 *days*). *Then sesquichloride of iron. Opiate enema on* 12*th day. Brandy to* 24 *oz.*

Typhus.—Mary H., æt. 14. Adm. Oct. 1. Disch. Oct. 31. In H. 30 days. *Recovery.* Dr. Johnson, vol. 18, p. 15. Prev. ill. 7 days. Shivering, pains in back and limbs, headache, vomiting, anorexia. Measly rash on abdomen and trunk on adm., with a few rose spots. Drowsiness, diarrhœa (11 days), and nocturnal delirium on 7th day. Tongue dry. Deafness. Trace of albumen about 9th day. Convalescent on 23rd day.

Dilute muriatic acid, chloric ether, and liquor cinchonæ. Opiate enemata. Brandy to 18 *oz.*

Typhus.—M. B., æt. 19, Weaveress. Adm. Oct. 14. Disch. Dec. 18. In H. 65 days *Recovery.* Dr. Garrod, vol. 3, p. 85. Nursed sister with typhus. Prev. ill. 2 days. Shivering and rapid prostration. On 3rd day, P. 120, R. 30. Indistinct rubeoloid rash. Very deaf. On 11th day tongue dry and brown. P. 140, R. 50. Motions passed under her on 14th day. Delirium on 3rd day (18 days). Urine albuminous on 12th day, and not quite free from albumen till 21st day. Convalescent about 40th day.

Dilute nitro-muriatic acid and chloric ether (24 *days*). *Then same with bark. Brandy to* 21 *oz. Turpentine stupes to back.*

Typhoid Fever.—Wm. H., æt. 16, Adm. Nov. 17. Disch. Dec. 9. In H. 22 days. *Recovery.* Dr. Johnson, vol. 19, p. 156. Prev. ill. 13 days. Shivering, headache, lassitude, diarrhœa. Tympanites, cœcal gurgling. Tongue red and glazed. P. 102, R. 24. Crepitation at right base. Moderate eruption. No spots after 21st day. Purging under three times in 24 hours, till 22nd day. Convalescent on 27th day.

Dilute nitro-muriatic acid, and bark (9 *days*). *Then quinine and iron. Brandy,* 8 *oz. Opiate enemata. Turpentine stupes.*

Typhoid Fever.—J. H., æt. 23, Clerk. Adm. Sept. 12. Disch. Sept. 28. In H. 16 days. *Recovery.* Dr. Johnson, vol. 16, p. 63. Prev. ill. 4 days. Shivering, headache, lassitude, loss of appetite. Tongue on 7th day dry, with central brown streak and red tip. P. 112. Rose spots appeared from 5th till 11th day. Purged under five times daily for some time.

Nitro-muriatic acid, chloric ether, and decoction of bark.

Typhoid Fever.—Maria P., æt. 25, Servant. Adm. Oct. 27. Disch. Nov. 21. In H. 25 days. *Recovery.* Dr. Johnson, vol. 18, p. 73. Prev. ill. 14 days. Shivering, loss of appetite, prostration, occipital headache. Tongue red and dry, slight cœcal tenderness. Tympanites. Scanty rose spot, eruption visible on

adm. Purged under five times daily, till 24th day. Convalescent about 36th day.

Dilute nitro-muriatic acid, chloric ether, and bark (21 *days*). *Cod-liver oil and syrup of iodide of iron. Opiate enemata. Brandy to* 12 *oz.*

Typhoid Fever.—J. H., æt. 19, Draper's Porter. Adm. Aug. 18. Disch. Sept. 12. In H. 25 days. *Recovery.* Dr. Garrod, vol. 1, p. 207. Indisposed. Then shivering, headache, lassitude, sore throat, loss of appetite. Tongue red, and glazed in centre. Ulcer on uvula. Slight cœcal tenderness. On 9th day, eight or ten abdominal rose spots. Purged under five times daily. Convalescent about 21st day.

Chloric ether and liquor ammoniæ acetatis (10 *days*). *Then chloric ether and liquor cinchonæ.*

Typhoid Fever.—E. N., æt. 25, Mirror Mounter. Adm. July 27. Disch. Sept. 23. In H. 38 days. *Recovery.* Dr. Garrod, vol. 1, p. 187. Prev. ill. 5 days. Shivering, headache, profuse purging before adm. On 8th day, P. 96, R. 28, 6—10 rose spots on abdomen. Slight cough, crepitus at bases. On 10th day, abdominal tenderness and cœcal gurgling. On 11th day, purged eight or nine times. Tongue dry, brown, and tremulous. P. 100. Purging ceased about 16th day. Convalescent on 21st day.

Acetate and carbonate of ammonia. Then nitric acid and bark. Brandy, 12 *oz. Opiate enemata.*

Typhoid Fever.—Charlotte H., æt. 24. Adm. Oct. 4. Disch. Dec. 12. In H. 69 days. *Recovery.* Dr. Johnson, vol. 17, p. 121. Prev. ill. 6 days. Headache, thirst, anorexia. Moderate diarrhœa. Scanty rose spot eruption, first seen on 6th day. P. 100, R. 24. Convalescent about 21st day. On 23rd day, phlegmasia-dolens of left leg (50 days).

Dilute nitro-muriatic acid, chloric ether, and bark (15 *days*). *Bark and carbonate of ammonia* (15 *days*). *Iron and quinine* (39 *days*). *Brandy,* 6 *oz. Morphia.*

Typhoid Fever?—Sarah P., æt. 37. Adm. Aug. 6. *Died* on Aug. 9. Dr. Johnson. Ill fed lately. Prev. ill. about 14 days. Headache, languor, delirious at times. On adm. skin hot, no eruption, but sudamina; stupor. P. 140, R. 40. Crepitation at bases; abdomen tympanitic; tongue dry and brown. Purged on 7th and 8th. Sloughing of nates. Died comatose on 9th.

Dilute nitro-muriatic acid, chloric ether, and liquor cinchonæ. Opiate enema. Brandy to 16 *oz.*

Typhoid Fever.—E. E., æt. 30, Probationer. Adm. July 17. Disch. Sept. 12. In H. 57 days. *Recovery.* Dr. Johnson, vol. 18, p. 11. Small-pox 21 years ago. Shivering on July 17th (1st day). On 4th day, P. 128, R. 32. Thick creamy fur on tongue. Slight cœcal tenderness about 12th day. Tympanites. Profuse diarrhœa. None after 22nd day.

Carbonate of ammonia and chloric ether. Then quinine and chloric ether. Wine, 4 *oz.*

Typhoid Fever.—Emma W., æt. 12½. Adm. Dec. 4. Disch. Dec. 30. In H. 26 days. *Recovery.* Dr. Johnson, vol. 17, p. 177. Prev. ill. 3 weeks. Shivering, anorexia, vomiting, diarrhœa, deafness, and delirium. On adm. P. 120. Purged 5 times in 24 hours, none after 24th day. A few rose spots on abdomen. Tongue clean. Slight delirium two nights. Convalescent on 35th day.

Dilute nitro-muriatic acid and decoction of bark. Opiate enemata (3). *Brandy,* 4 *oz.*

Typhoid Fever.—Alexr. F., æt. 8. Adm. June 25. Disch. July 22. In H. 27 days. *Recovery.* Dr. Beale, vol. 6, p. 208. Prev. ill. 8 days. Shivering, vomiting, purging. On 10th day one rose spot, wheezing, dry cough. On 11th

day, P. 130, R. 56. Tongue red, and dry at tip, edges yellow. Purging under 8 times daily, ceasing on 18th day. No fresh spots after 14th day. Convalescent on 26th day.

Dilute nitro-muriatic acid, liq. cinchonæ, and chloric ether (3 *days*). *Sp. ammon. arom. and eth. chlor.* (11 *days*). *Then dilute muriatic acid and quassia. Opiate enemata. Pepsine.*

Typhoid Fever.—Disease of Gall Bladder.—Eleanor M., æt. 53. Adm. June 10. *Died* on Sept. 6. In H. 88 days. Dr. Johnson, vol. 15, p. 243. Frequent vomiting 4 months. Loss of appetite, flesh, and strength, 2 months. Sour risings, waterbrash, and pain after food. Passed some blood in motions 3 weeks ago. Tender moveable tumour in right hypochondrium, just below margin of ribs. Aug. 22.—P. 120. Swelling and redness of elbows. Free sweating. 9 days later very weak, tongue brownish. P. 128. 4 days later delirious. P. 140, R. 36. Tongue brown and dry. Purged. Abdomen tender and tympanitic, with two or three rose spots on it. Unconsciousness and death 2 days later.—P.M.—Gall bladder as large as a goose's egg, containing greenish semifluid matter, consisting of pus, granular corpuscles, and nucleated cells. Walls very thick, containing plates of chloresterine. Cystic duct blocked by a gall stone.

Opium. Purgatives. Sp. ammon. arom. Hydrocyanic acid. Ammonia, ether, and bark. Brandy, 12 *oz.*

Scarlet Fever.—M. R., æt. 21, Servant. Adm. July 3. *Died* on July 8. In H. 5 days. Dr. Johnson, vol. 17, p. 73. Had been exposed to contagion. Prev. ill. 2 days. Constant vomiting after first day. Rash on 3rd day. Throat sore. Tonsils red. Much dysphagia. Tongue creamy in centre, red at tip and edges; afterwards red and glazed. Delirious on 3rd and 4th nights. On 6th day wrists painful and swollen, then knees and ankles. Death on 8th day. P.M.—Lungs somewhat gorged. A few tubercles in right apex. Advanced fatty degeneration of heart.

Quinine, chlorate of potass, and sesquichloride of iron. Iron and glycerine to throat.

Scarlet Fever.—E. R., æt. 11, School Girl. Adm. Sept. 28. Disch. Nov. 24. In H. 57 days. *Recovery.* Dr. Beale, vol. 5, p. 302. Caught at school. Prev. ill. 1 day. Rash fully out on adm. Tongue coated with thick, brown fur, red at tip and edges. P. 120. Tonsils and uvula red and swollen. Eyelids puffy on 7th day. Desquamation began on 9th day. On 23rd day albumen in urine (21 days). On 39th day dulness and bronchial breathing at left base (2 days).

Sesquichloride of iron. Bark and dilute muriatic acid. Chloric ether and liquor ammoniæ acetatis. Hot air baths. Iron and glycerine to throat.

Scarlet Fever.—T. L., æt. 21, Tailoress. Adm. July 10. Disch. Aug. 8. In H. 29 days. *Recovery.* Dr. Beale, vol. 6, p. 230. No evidence of contagion. Prev. ill. 3 days. Shivering. Severe sore throat on 2nd day. Eruption on 4th day. Tongue creamy. Fauces red. Tonsils swelled, covered with yellow sloughs. On 11th day knees and wrists swollen and painful (10 days). Free sweating. Desquamation about 22nd day. Systolic bruit at apex on 23rd day. Highest pulse, 132.

Quinine, iron, and chlorate of potass (6 *days*). *Bicarbonate of potass and acetate of ammonia* (4 *days*). *Then bark, ether, and ammonia. Opiates.*

Scarlet Fever?—H. B., æt. ?, Hospital Nurse. Adm. Aug. 4. *Died* on Aug. 26. In H. 22 days. Dr. Beale, vol. 5, p. 248. Scarlet fever in next ward. Pain in chest, headache, and vertigo, 14 days. 4 days ago sore throat, pain in chest, purging, and vomiting (8 days). On adm. headache, tongue red and raw, whitish tonsillar exudation (till 18th day). Cuticle of arms desquamating. On 11th day urine albuminous, friction sound in front of left lung. From 13th to 17th day much crepitation over bases; friction sound very loud. On 23rd day

front of chest free from rales. Tympanites greater. Vomiting frequent. P. thready and rapid. Death on 27th day. No P.M.

Chlorate of potass, liquor cinchonæ and dilute muriatic acid. Ammonia and ether. Brandy to 12 *oz. Enemata of quinine and opium, and rue. Iron and glycerine to throat.*

Scarlet Fever.—C. S., æt. 13. Adm. Sept. 28. Disch. Dec. 9. In H. 72 days. *Recovery.* Dr. Beale. vol. 6, p. 290. Prev, ill. 3 days. Rash out on 2nd day. Slight sore throat. Desquamation began on 11th day. Face œdematous on 18th day. Urine albuminous on 22nd day (44 days). Mumps about 48th day. Began to improve about 27th day.

Sesquichloride of iron and dilute muriatic acid. Liq. ammoniæ acetatis and bark. Cod-liver oil. Purgatives. Hot air baths.

Scarlet Fever.—J. W., æt. 16, Clerk. Adm. Sept. 25. Disch. Oct. 10. In H. 15 days. *Recovery.* Dr. Johnson, vol. 17, p 108. Prev. ill. 3 days. Sore throat and dysphagia on 2nd day. Eruption on 3rd. Desquamation about 8th day.

Liq. ammon. acetatis and sp. etheris nitrici. Then sesquichloride of iron.

Scarlet Fever.—Mary A. T., æt. 13. Adm. June 11. Disch. July 1. In H. 20 days. *Recovery.* Dr. Garrod, vol. 1, p. 139. Prev. ill. 9 days. On adm. tonsils swelled, red, and ulcerated. Rash on day after adm., and left knee, ankles, and left wrist painful and swelled (5 days). Tongue red. Desquamation commenced about 10th day after adm. (20th day of illness).

Acetate of ammonia (7 *days*). *Then sesquichloride of iron.*

Scarlet Fever.—F. K., æt. 19, Goldmelter. Adm. June 6. Disch. July 11. In H. 35 days. *Recovery.* Dr. Johnson, vol. 15, p. 226. No evidence of contagion. Prev. ill. 3 days. Sore throat and shivering. Eruption on third day. Superficial ulceration of tonsils. Delirious on 4th, 5th, and 6th nights. Desquamation on 12th day. Abscess in right tonsil about 18th day.

Carbonate of ammonia and liq. ammon. acetatis (2 *days*). *Then chlorate of potass, quinine, and iron. Glycerine and sesquichloride of iron to throat.*

Scarlet Fever.—George W., æt. 10. Adm. Oct. 9. *Died* Oct. 14. In H. 5 days. Dr. Beale, vol. 5, page 292. Prev. ill. 1 day. Sore throat. Feverish and scarlet on 2nd day. On 3rd day, P. 140, R. 32. Swelling and superficial ulceration of tonsils. Diarrhœa on 4th day. On 6th day coarse crepitus over lungs. On 7th day comatose; motions passed under him. False membranes on fauces. Death on 8th day.

Chloric ether and liquor ammoniæ acetatis. Brandy. Nutrient enemata. Iron and glycerine to throat.

Scarlet Fever.—E. R., æt. 24, Probationer. Adm. Aug. 28. Disch. Sept. 23. In H. 26 days. *Recovery.* Dr. Beale, vol. 5, p. 266. Prev. ill. 3 days. Shivering. Rash out on 3rd day. Fauces red. Slight exudation on left tonsil. P. 120. Tongue furred.

Quinine, sesquichloride of iron, and chloric ether. Glycerine and sesquichloride of iron to throat.

Scarlet Fever.—Elizabeth G., æt. 5. Adm. Nov. 23. Disch. Dec. 5. In H. 12 days. *Recovery.* Dr. Beale, vol. 7, p. 17. Scarlet fever in surgical ward from which she was transferred. Fretful on 22nd, rash out next day. Fauces red. Desquamation commenced about Nov. 30.

Liquor ammoniæ acetatis, and chloric ether.

Scarlet Fever.—J. M., æt. 13, School Girl. Adm. Oct. 6. Disch. Nov. 12. In H. 37 days *Recovery.* Dr. Beale, vol. 5, p. 312. Other case from same school. Prev. ill. 2 days. Sore throat and loss of appetite. Rash well out on third day. P. 124. Fauces red. White patch on right tonsil. Desquamation on 9th day (23 days).

Liq. ammon. acetatis, sp. ammon. aromat., and chloric ether (9 *days*). *Then tonics. Brandy to* 4 *oz.*

Scarlet Fever.—S. S., æt. 10, School Girl. Adm. Oct. 6. Disch. Oct. 26. In H. 20 days. *Recovery.* Dr. Garrod, vol. 3, p. 61. Caught at school. Prev. ill. 8 days. Rash on 2nd day. Tonsils red and swelled. Tongue white, with red tip and edges. P. 152. Desquamation began about 7th day.

Liquor ammoniæ acetatis. Iron and glycerine to throat.

Scarlet Fever.—E. L., æt. 19¾, School Girl. Adm. Sept. 26. Disch. Oct. 17. In H. 21 days. *Recovery.* Dr. Johnson, vol. 17, p. 106. Prev. ill. 1 day. Vomiting. Sore throat on 2nd day. Rash fully out on adm. Tonsils red and swollen. Desquamation began about 6th day.

Sesquichloride of iron.

Scarlatina Puerperalis.—Mary A. F., æt. 29, married. Adm. June 20. Disch. July 18. In H. 28 days. *Recovery.* Dr. Garrod, vol. 2, p. 185. Confined on 13th. Then languor and loss of appetite. Shivering on 16th, and eruption same day (9 days). Severe vomiting and diarrhœa on 17th and 18th, Delirious on night of 19th. Tongue, fauces, and tonsils red. Intense marked eruption. P. 124. Desquamation about 27th. No rash on child.

Liq. ammon. acetatis, aromatic spts. of ammonia, and chloric ether—changed for sesquichloride of iron on 13th.

Erysipelas of Arm.—J. O., æt. 36, Compositor. Adm. Sept. 26. Disch. Oct. 12. In H. 16 days. *Recovery.* Dr. Johnson, vol. 2, p. 3. Had been living badly. Prev. ill. 6 days. Shivering and cramps in limbs. Next day swelling and redness about right elbow, down forearm and hand 5 days later. Suppuration about elbow 2 days later.

Sesquichloride of iron, quinine, and chloric ether. Brandy 2 oz. Purgatives. Morphia. Abcess opened.

Erysipelas of Face.—J. F., æt. 18, Shoemaker. Adm. Sept. 8. Disch. Sept. 19. In H. 11 days. *Recovery.* Dr. Garrod, vol. 2, p. 277. Erysipelas of scalp 3 years ago. Three months was in K.C.H., under Dr. Johnson, with erysipelas of face. Prev. ill. 2 days. Commenced on bridge of nose, and extended over upper part of face and backwards to ears. P. 88.

Calumba and sesquichloride of iron. Beer 1 pt. Collodion and oil locally.

Erysipelas of Face.—J. F., æt. 18, Shoemaker. Adm. Oct. 4. Disch. Oct. 21. In H. 17 days. *Recovery.* Dr. Johnson, vol. 17, p. 124. Fourth attack of erysipelas. Prev. ill. 1 day. Began on upper lip. All over face on adm. On back of scalp 3 days later.

Quinine, chloric ether, and sesquichloride of iron. Brandy 4 oz. Collodion and oil locally.

Erysipelas of Face.—F. B., æt. 17, Compositor. Adm. Sept. 12. Disch. Sept. 26. In H. 14 days. *Recovery.* Dr. Beale, vol. 6, p. 246. First attack. Caught from father. Prev. ill. 2 days. Shivering, nausea, and vomiting. Began on nose. Cheeks vesicated. Most of face and front of scalp afterwards affected.

Calumba and sesquichloride of iron. Brandy 3 oz. Collodion and oil locally.

Erysipelas of Face.—Mary A. W., æt. 48, Cook. Adm. Sept. 22. Disch. Oct. 5. In H. 13 days. *Recovery.* Dr. Garrod, vol. 3, p. 44. First attack. Prev. ill. 1 day. Preceded by severe toothache. Commenced near right eye. On adm. general redness and swelling of face, eyes nearly closed. 4 days later convalescent, but relapsed next day, and again 6 days later.

Calumba and sesquichloride of iron. Aperients. Collodion and oil locally.

Erysipelas of Face.—Margaret A., æt. 19, married. Adm. Oct. 3. Disch. Oct. 17. In H. 14 days. *Recovery.* Dr. Garrod, vol. 3, p. 50. Got wet 2 days ago. Next day erysipelas began about left eye, and soon spread all over face. Vesications on forehead. P. 128. Well 10 days after adm.

Sesquichloride of iron. Collodion and castor oil locally.

Erysipelas of Face.—J. P., æt. 24, Engraver. Adm. Oct. 26. Disch. Nov. 7. In H. 12 days. *Recovery.* Dr. Garrod, vol. 3, p. 112. Ill fed lately. Five days ago, shivering, vomiting, and headache. Two days later erysipelas began in cheeks. Four days later face much swelled, eyes closed, slight extension to scalp, vesication, on left cheek P. 92.

Opiates. Brandy 6 oz. Collodion and oil to face. Afterwards iron.

Erysipelas of Face.—Sarah W.. æt. 53. Adm. July 16. Disch. Aug. 12. In H. 27 days. *Recovery.* Dr. Johnson, vol. 18, p. 1. First attack. Vomiting and loss of appetite, 3 weeks. Prev. ill 5 days. Spread from left eye over whole face and scalp. On adm. almost unconscious. P. 128, thready Tongue dry and brown.

Quinine and sesquichloride of iron. Brandy to 12 oz. Collodion and oil locally.

Erysipelas of Face.—J. McM., æt. 60, Reporter. Adm. July 7. *Died* on July 7. Dr. Beale. No history. Moribund when admitted.

Brandy. Sesquichloride of iron and chloric ether. Collodion and oil locally.

Variola.—Mary A. L., æt. 27. Adm. July 27. Disch. July 28. In H. 1 day. Dr. Johnson.

Chloric ether and liq. ammon. acetatis. Castor oil.

Variola.—Eliza L., æt. 19. Adm. July 3. Disch. July 4. In H. 1 day. Dr. Johnson.

Variola.— G., Student. Adm. Nov. 16. Disch. Nov. 17. In H. 1 day, Dr. Johnson.

Febricula.—Sophia B., æt. 15. Adm Oct. 13. Disch. Oct. 24. In H. 11 days. *Recovery.* Dr. Beale, vol. 5, p. 314. Prev. ill. 1 day. Headache. Diarrhœa. Furred tongue. P. 128.

Chloric ether and liq. ammon. acetatis (4 days). Then quassia and muriatic acid.

Febricula.—Ruth G., æt. 24, married. Adm. Nov. 6. Disch. Dec. 9. In H. 33 days. *Recovery.* Dr. Garrod, vol. 4, p. 8. Prev. ill. 5 days. P. 104. R. 22. Slight dulness over right lower lobe, and at left base. Crepitus over lower lobes of both lungs. Convalescent on 21st day.

Bark and dilute nitro-muriatic acid. Brandy 6 oz. Castor oil.

Febricula.—E. C , æt. 22, Servant. Adm. Oct. 31. Disch. Oct. 21. In H. 8 days. *Recovery.* Dr. Beale, vol. 6, p. 314. Prev. ill. 4 days.

Castor oil. Chloric ether and sal-volatile. Citrate of iron.

Febricula.—Eliza H., æt. 13. Adm. Oct. 3. Disch. Oct. 14. In H. 11 days. *Recovery.* Dr. Beale, vol. 6, p. 298. Prev. ill. 5 days. Headache, sore-throat, nausea, languor, cough, discharge from ears.

Liq. ammon. acetatis, chloric ether, henbane, and bark. Ears syringed.

Febricula.—Ann B., æt. 19, Servant. Adm. Nov. 26. Disch. Dec. 5. In H. 9 days. *Recovery.* Dr. Johnson, vol. 17, p. 169. Prev. ill. 1 day. Well 6 days later.

Chloric ether and liq. ammon. acetatis. Iron and quinine.

Febricula.—E. F., æt. 32, Hospital Nurse. Adm. July 28. Disch. Aug. 5. In H. 8 days. *Recovery.* Dr. Johnson, vol. 17, p. 63. Prev. ill. 1 day. Sore throat, shivering, nausea, constipation. Well on 8th day.

Chloric ether and liq. ammon. acetatis. Quinine and iron. Aperients.

Febricula.—Cath. G., æt. 6. Adm. June 29. Disch. July 11. In H. 12 days. *Recovery.* Dr. Johnson, vol. 17, p. 3. Prev. ill. 3 days. Headache, vomiting,

purging, slight sore throat. P. 120. Tongue furred, wheezing at bases of lungs
Castor oil. Sesquichloride of iron.

Ague.—J. D., æt. 22, Labourer. Adm. July 29. Disch. Aug. 15. In H. 17. days. *Recovery.* Dr. Johnson, vol. 17, p. 80. Prev. ill. 7 weeks. Tertian. Pain over spleen 6 weeks. No perceptible enlargement. Very anæmic. Had a paroxysm on 2nd day after adm. None afterwards.
Inf. quassia (2 days). Then quinine. Afterwards quinine and sesquichloride of iron.

Ague.—Ellen G., æt. 39, Servant. Adm. May 26. Disch. July 8. In H. 43 days. *Recovery.* Dr. Beale, vol. 2, p. 140. Back from India 7 weeks. While there had several attacks of fever. Cough since return. Irregular ague 3 weeks. Tongue foul; breath fœtid; bowels confined. Liver tender. P. 140, R. 32. Systolic bruit at apex of heart. Lungs healthy. No shivering after May 28th.
Quinine and sulphuric acid. Quinine and sulphate of iron.

Ague.—Ellen G., æt. 39, Servant. Adm. Oct. 13. Disch. Oct. 24. In H. 11 days. *Relieved.* Dr. Johnson, vol. 18, p. 37. Three or four aguish attacks since disch. last July. Cough troublesome. Harsh breathing and click at apex of left lung. Ague on 15th, 17th, and 19th. None afterwards.
On Oct. 19, quinine and sulphuric acid.

Muscular Atrophy.—J. L., æt. 30, Coal Miner. Adm. June 9. Disch. June 20. In H. 11 days. *Unrelieved.* Dr. Beale, vol. 6, p. 176. Wasting of right shoulder muscles, 2 years, consequent on severe bruise of shoulder by falling coal 3 years ago. Now right deltoid and supra and infra spinatus quite gone, right biceps very small. Corresponding loss of motor power.
Galvanism.

Muscular Atrophy.—E. W., æt. 29, Gold Digger. Adm. Oct. 15. Disch. Oct. 24. In H. 9 days. *Relieved.* Dr. Johnson, vol. 19, p. 113. Wasting of muscles of right arm and hand, especially those supplied by ulvar nerve. Numbness and loss of power in arm. Came on suddenly 2¾ years ago, with cerebral oppression, and has increased since. Similar attack 7 years ago, preceded by pain in right shoulder. Face slightly drawn to the right. Memory impaired.
Shower bath. Frictions. Galvanism. Strychnia and sesquichloride of iron.

Muscular Atrophy.—T. T., æt. 16, Sailor. Adm. June 1. Disch. Aug. 1. In H. 61 days. *Nearly well.* Dr. Johnson, vol. 15, p. 218. Contraction of right hand. Muscles of right hand and forearm much wasted, especially the extensors. Came on after acute rheumatism a year ago.
Quinine and sesquichloride of iron. Galvanism. Cold splash.

Muscular Atrophy.—Rebecca P., æt. 15, Servant. Adm. Feb. 4. Disch. July 4. In H. 150 days. *Recovery.* Dr. Johnson, vol. 16, p. 105. Eight weeks ago pain in left biceps, extending up and down arm, followed by numbness, loss of motion and sensation, and contraction of flexors. Six weeks ago, pain in left calf, followed by rigidity of leg, drawing inwards of foot, and complete loss of motion. Tenderness of cervical and upper dorsal spine.
Iodide of potassium. Quinine and sesquichloride of iron. E. L. and emp. bellad. to spine.

Muscular Atrophy.—L. S., æt. 35, Cook. Adm. Nov. 4. Disch. Dec. 2. In H. 28 days. *Relieved.* Dr. Johnson, vol. 18, p. 79. Prev. ill. 2 years. Pain at times in left side of neck. Head drawn to left side. Is obliged to support it with left hand. Atrophy of left sterno mastoid and right upper border of trapezius.
Iron and gentian. Shower baths. Aperients.

Muscular Atrophy.—E. B., æt., 19, Housemaid. Adm. Sept. 18. Disch.

Nov. 25. In H. 68 days. *Improved.* Dr. Johnson, vol. 19, p. 77. Extreme atrophy of left sterno-mastoid 9 months. Head drawn to the right. Upper border of left trapezius wasted.

Iodide of potassium, bicarbonate of potass and bark. Galvanism.

Hysteria.—M. H., æt. 20, Housemaid. Adm. July 1. Disch. July 15. In H. 14 days. *Relieved.* Dr. Johnson, vol. 15, p. 269. Ailing 14 months. Frequent hysterical fits 3 weeks ago. Headache, loss of appetite, and anœmia.

Valerianate of zinc. Quinine and sesquichloride of iron.

Hysteria.—G. F., æt. 28, Tailor. Adm. July 15. Disch. July 22. In H. 7 days. *Recovery.* Dr. Garrod, vol. 2, p. 230.

Bark and carbonate of ammonia. Asafœtida. Purgatives.

Hysteria.—Jane S., æt. 40, Married. Adm. July 17. Disch. Aug. 8. In H. 22 days. *Recovery.* Dr. Garrod, vol. 1, p. 179. Acute rheumatism 3 years ago. Menorrhagia 3 months. Prev. ill. 4 days. Attack resembling ac. rh., but no swelling or redness of joints. Did not improve till Aug. 3.

Liq. ammon. Acetat. Liquor potassæ. Asafœtida after Aug. 1.

Hysteria.—Jane E., æt. 17, Servant. Adm. May 28. Disch. July 4. In H. 37 days. *Unrelieved.* Dr. Garrod, vol. 2, p. 146. Prev. ill. 6 days. Headache. Pain and tenderness over crest of right ilium. Obstinate constipation.

Asafœtida. Purgatives. Sulphate of iron.

Hysteria.—Sarah G., æt. 20. Adm. Dec. 14. Disch. Dec. 23. In H. 9 days. *Relieved.* Dr. Johnson, vol. 19, p. 185. Vomiting and diarrhœa 3 weeks ago. Tenderness of lower dorsal vertebræ; umbilical pain; furred tongue; constipation.

Asafœtida. Calomel and colocynth. Shower baths.

Hysteria.—S. B., æt. 31, Hospital Nurse. Adm. July 16. Disch. Sept. 24. In H. 70 days. *Recovery.* Dr. Beale, vol. 5, p. 232. Lately pain in joints, headache, and constipation. Delirium on day after adm. (3 days). Acute rheum. 11 days later (49 days). Heart unaffected.

Liq. ammon. acetatis. Then valerian, chloric ether, and ammonia. Bicarbonate and nitrate of potass. Opium and quinine for rheum. Bark and valerian. Bark and asafœtida. Shower baths.

Hysteria.—M. A. S., æt. 31, Married. Adm. Aug. 22. Disch. Sept. 1. In H. 11 days. *Unrelieved.* Dr. Beale, vol. 6, p. 238. Lost her voice twice 2 years ago. Prev. ill. 9 months. Pain in limbs and head. Giddiness. Dorsal spine very tender. Occasional flatulence. Disch. at her own request.

Valerian, aromatic spts., ammonia, and mist. camph.

Hysteria.—M. B., æt. 20, Laundress. Adm. Oct. 30. Disch. Nov. 13. In H. 14 days. *Recovery.* Dr. Johnson, vol. 19, p. 132. Lost her voice twice about a year ago. Three days ago became speechless, after an hysterical fit. Voice returned suddenly on 4th day after adm. Menorrhagia.

Chlorate of potass, sesquichloride of iron and quassia. Iron, alum, and henbane.

Hysteria.—Eleanor A., æt. 37, Married. Adm. Oct. 2. Disch. Oct. 14. In H. 12 days. *Recovery.* Dr. Johnson, vol. 18, p. 29. Hæmatemesis thrice last year. Weakness of voice 3 weeks. Very nervous.

Bark, aromatic spts. of ammonia, and chloric ether. Asafœtida. Black draught.

Acute Rheumatism.—Chorea.—J. L., æt. 14, Light Porter. Adm. June 24. Disch. Aug. 1. In H. 38 days. *Recovered nearly.* Dr. Garrod, vol. 1, p. 155. Got wet 5 weeks ago. Three days later acute pain in ankles, then in knees, shoulders, wrists, and chest. Chorea began 3 weeks ago, in left leg, then in left

arm, now general; left side worst; face drawn to the right. Tongue furred, jerked back. Loud systolic bruit over heart's apex. Free from pain on 13th day after adm.

Quinine and bicarbonate of potass (6 *days*). *Then quinine, sulphate of iron, and dilute sulphuric acid.*

Rheumatic Pericarditis.—Chorea.—Thomas S., æt. 7. Adm, June 22. Disch. July 29. In H. 37 days. *Recovery.* Dr. Johnson, vol. 16, p. 255. Got wet a month ago. Pain in neck, chest, right arm, and leg 3 weeks. Chorea of right arm, leg, and face, 10 days. On adm. P. 150, R. 28. Cardiac dulness increased. Loud pericardial to and fro sound (25 days). Systolic bruit at heart's apex. Crepitation at bases of lungs. Choreic over 35 days. Free from pain on 5th day after adm.

Quinine, opium, and carbonate of ammonia. Afterwards quinine and sesquichloride of iron.

Chorea.—Ellen K., æt. 10. Adm. June 9. Disch. July 4. In H. 26 days. *Much relieved.* Dr. Johnson, vol. 17, p. 61. Chorea from fright 2 years ago (12 months). Was frightened 9 weeks ago. Choreic since. Face and arms affected first. Left side worst. Slight attack.

Quinine, iron, and splash baths.

Chorea.—Sarah L., æt. 13, Servant. Adm. Nov. 2. Disch. Dec. 23. In H. 51 days. *Recovery.* Dr. Garrod, vol. 1, p. 225. First attack. Frightened. Prev. ill. 1 month. In right arm first, and more in right side than in left. Lower dorsal spine tender.

Asafœtida (19 *days*). *Shower baths and mist. camph.* (26 *days*). *Sulphate of iron and shower baths* (6 *days*).

Chorea.—M. B., æt. 22, married, Needlewoman. Adm. Oct. 14. Disch. Dec. 5. In H. 52 days. *Relieved.* Dr. Beale, vol. 5, p. 316. First attack. No cause. Thick speech, and occipital headache 10 months. Movements 7 months, first in right arm and leg. Now much jerking of trunk and limbs. Recovered nearly.

Sesquichloride of iron. Purgatives. Cod-liver oil.

Chorea.—John S., æt. 38. Adm. Nov. 30. Disch. Dec. 1. In H. 1 day. Dr. Beale. Went out of hosp.

Chorea.—C. S., æt. 20. Adm. Oct. 17. Disch. Oct. 24, at her own request. In H. 7 days. *Unrelieved.* Dr. Johnson, vol. 18, p. 47. Chronic chorea of right side, 8 years, preceded by rheumatic pains in same side from sleeping in damp bed. Has had treatment.

Quinine, iron, and calumba. Shower baths.

Epilepsy.—Renal Disease.—H. P., æt. 18, Farm Labourer. Adm. Nov. 17. *Died* Nov. 22. In H. 5 days. Dr. Beale, vol. 6, p. 335. Ill 5 years with œdema of legs, occasional epileptic fits, and constant vomiting. Now very pallid; urine very albuminous and abundant. Systolic bruit at heart's apex. Frontal headache. Epistaxis. Constant vomiting. Six fits while in hosp., death during last. P.M.—Serous effusion into arachnoid. Slight atheroma of basic cerebral arteries. A portion of under surface of left cerebellar lobe, yellow and softened. Similar softening at posterior part of right cerebral hemisphere. Heart, 20½ oz. Left vent. hypertrophied. Aortic valves thickened. Atheromatous patches in mitral valve. Kidneys pale and contracted.

Dilute hydrocyanic acid and effervescing mixture. Bark, soda, rectified spt., and creosote.

Epilepsy.—J. H., æt. 47, Farrier. Adm. Oct. 13. Disch. Nov. 9. In H. 27, days. *Much relieved.* Dr. Beale, vol. 6, p. 306. Left hemiplegia 2 years ago

(3 months). Prev. ill. 1 year. Headache, epileptic fits, and gradual loss of power in left arm and leg. Fits preceded by aura from left hand, double vision, and spectral illusions. Had four attacks while in hospital.

Purgatives. Dilute muriatic acid and liq. cinchonæ. E. L. nuchæ.

Epilepsy.—Jane W., æt. 23, Married. Adm. Nov. 25. Disch. Dec. 5. In H. 10 days. *Relieved.* Dr. Johnson vol. 18, p. 115. Eleven months ago had 3 fits in 3 weeks. Since then "fainting fits." Profuse catam. flow 2 weeks back. Epileptic 1 week. Pallid. Basic systolic bruit and venous murmur in neck. No fits in hosp.

Quinine and iron. Opiates. Castor oil.

Epilepsy.—S. F., æt. 58, Blind Painter. Adm. July 29. Disch. Aug. 12. In H. 14 days. *Relieved.* Dr. Garrod. Epileptic 16 years. Caused by a blow on the head (fall). Occasional headache. Mind somewhat affected. Had 3 fits in last 2 months. None while in hosp.

Iodide of potassium and sulphate of magnesia.

Epilepsy.—J. F., æt. 34, Billposter. Adm. Aug. 6. Disch. Aug. 8. In H. 2 days. *Relieved.* Dr. Johnson. Intemperate. Struck head in a fall 29 years ago. A fit 2 years afterwards. Now epileptic 2 years. Fit on Aug. 6, with transient palsy of left face, arm, and leg (2 days).

Sp. ammon. arom. Haust. Sennæ Co.

Epilepsy.—Harriet P., æt. 18, Servant. Adm. July 28. Disch. Aug. 15. In H. 18 days. *Unrelieved.* Dr. Garrod, vol. 1, p. 185. Hereditary. Prev. ill. 14 days. Two fits daily before adm. Headache. Faint systolic bruit at apex of heart. Six fits while in hosp.

Asafœtida. Oxide of silver.

Epilepsy.—Emma D., æt, 19, Cook. Adm. Sept. 19. Disch. Sept. 30. In H. 11 days. *Recovery.* Dr. Garrod, vol. 3, p. 27. Catamenia every fortnight, 18 months. Headache 8 months, worse last week. Fell and struck head 14 days ago. Epileptic fits during last 4 days. Fits preceded by an aura from right great toe to parietal region.

Purgatives.

Delirium.—T. P., æt. 45, Brewers' Man. Adm. July 10. Disch. July 14. In H. 4 days. Dr. Garrod, vol. 2. p. 226. Had been nursing a delirious patient. Became violent and delirious on night but one before adm. Quite well since, but frightened.

Delirium Tremens.—J. G., æt. 30, Waiter. Adm. Sept. 28. Disch. Oct. 7. In H. 9 days. *Recovery.* Dr. Beale, vol. 5, p. 306. First attack. Embarassed. Intemperate. Prev. ill. 6 days. Tremulous and troublesome. Slight spectral illusions. Convalescent on 3rd day after adm.

Opiates. Then liq. cinchonæ and dilute muriatic acid.

Delirium Tremens.—H. C.. æt. 32, Baker. Adm. Aug. 4. Disch. Aug. 8. In H. 4 days. *Recovery.* Dr. Johnson. Fourth attack. Drinks freely. Has had much trouble. Lately nervous and sleepless. Now very tremulous. Tongue furred. Has spectra. Delirious for two days after adm.

Chloric ether and carbonate of ammonia. Opiates.

Delirium Tremens.—H. C., æt. 32, Baker. Adm. Sept. 21. Disch. Sept. 28. In H. 7 days. *Recovery.* Dr. Beale, vol. 6, p. 268. Fifth attack. Had been drinking before adm. Prev. ill. 3 days. Spectra. Tremors. Delirium. Sleeplessness. Noisy on 2nd day after adm.

Opiates. Antimony. Aperients.

Delirium Tremens.—S. G., æt. 28, Harness Maker. Adm. July 13. Disch. Aug. 4. In H. 18 days. *Recovery.* Dr. Johnson, vol. 16, p. 273. Third attack.

Had been drinking a week. Ill 2 days. Spectra. Sleeplessness. Considerable tremor. Remained tremulous for a fortnight.

Chloric ether, carbonate of ammonia, quinine, and opium. Afterwards quinine and strychnine.

Delirium Tremens.—E. W., æt. 33, Cellarman. Adm. July 9. Disch. July 18. In H. 9 days. *Recovery.* Dr. Johnson, vol. 15, p. 271. Drinks freely. Prev. ill. 4 days. Sleepless. No tremor, but movements hurried. Fancies a wine-peg is sticking in his left hand. Convalescent on 4th day after adm.

Chloric ether and carbonate of ammonia. Quinine and opium. Afterwards quinine and sulphuric acid.

Delirium Tremens.—E. B., 43, Carpenter. Adm. Oct. 15. Disch. Oct. 21. In H. 6 days. *Recovery.* Dr. Beale. First attack 4 years ago. Had been drinking before adm. Spectra. Slight tremor. P. 106.

Opiates. Brandy. Aperients. Bark and carbonate of ammonia.

Delirium Tremens.—Dan. S., æt. 40. Adm. Dec. 1. Disch. Dec. 4. In H 3 days. *Recovery.* Dr. Johnson, vol. 19, p. 171. Drinks. Has had anxiety lately. Jumped out of window in a fright 3 days ago. Now tremulous and sleepless, with spectral illusions.

Opiates.

Delirium Tremens.—J. P., æt. 26, Engineer. Adm. Nov. 5. Disch. Nov. 7. In H. 2 days. *Recovery.* Dr Johnson, vol. 19, p. 138. First attack. Drinking 3 days. Prev. ill. 1 day. Sleep disturbed. Slight spectra. Tongue furred. Bowels confined. No tremor. P. 52.

Opiates. Brandy. Quinine.

Delirium Tremens.—Jane B., æt. 45. Adm. Nov. 6. Disch. Nov. 21. In H. 15 days. *Recovery.* Dr. Johnson, vol. 19, p. 145. Very intemperate. Third attack. Drunk 4 days. Prev. ill. 2 days. Talkative, tremulous, and sleepless. Eyes red. Is surrounded by rats. No improvement for a week. Bronchitis.

Opium. Gin. Calomel and colocynth. Quinine, sulphuric acid, chloric ether and squills. Turpentine stupes.

Dementia.—Sarah T., æt. 60. Adm. Dec. 8. Disch. Dec. 29. In H. 21 days. *Unrelieved.* Dr. Beale, vol. 8, p. 13. Memory failing 12 months. Ill fed some time. Pain in right side of head, and constipation, 3 weeks. Now emaciated, delirious at times; refuses food; passes water under her. Bowels confined.

Quinine and dilute sulphuric acid. Opium. Purgatives.

Dementia.—George J., æt. 18. Adm. Sept. 12. Disch. Sept. 15. In H. 3 days. *Unrelieved.* Dr. Johnson.

Bark, opium, and carbonate of ammonia.

Dementia.—George H., æt. 15. Adm. Sept. 10. Disch. Oct. 28. In H. 48 days. *Unrelieved.* Dr. Garrod, vol. 3, p. 2. Gradual alteration in habits for last 8 months. Now sallow, sulky, and stupid, with tumid abdomen and large appetite.

Purgatives. Galvanism. Shower baths. Asafœtida. Iron.

Cerebral Softening.—R. B., æt. 57, Leather Dresser. Adm. May 12. Disch. May 14. In H. 2 days. *Unrelieved.* Dr. Johnson, vol. 16, p. 203. Ill 2 years. Memory impaired. Speech thick and rambling. Arms shaky. Unable to pick up a pin with left hand. Tongue protruded to the right (3 weeks). Radial arteries rigid and tortuous. Heart feeble.

General Paralysis.—J. R., æt. 39, Cabman. Adm. Sept. 16. Disch. Oct. 31. In H. 45 days. *Relieved.* Dr. Johnson, vol. 19, p. 66. Had syphilis 20

years ago. Gradual weakness of left leg 4 years, and of right leg 4 months. Twitchings of legs 4 months. Hands numb 2 months. Occasional incontinence of fæces and urine. Increase of palsy during last 4 days, sensation diminished. Left arm slightly weak. Sense of constriction round abdomen. Frontal headache; memory impaired. Has psoriasis palmaris.

Bichloride of mercury, iodide of potassium, and bark. Similar treatment for 5 weeks before adm.

FACIAL PALSY.—R. P., æt. 45, House Painter. Adm. Sept. 14. Disch. Oct. 28. In H. 44 days. *Recovery.* Dr. Garrod, vol. 3, p. 12. At trade 4 years; never suffered from lead. Three weeks ago cold wind on right side of face during day's work. Soon afterwards pain in right, head, neck, and face, and lately in right ear. Three days ago palsy of parts supplied by rt. portio dura. Tongue deviates to the left. Free from pain on 19th day after adm. Nearly well 5 days later.

Iodide of potassium, with bicarbonate of potass, and then with bark. Blisters and sinapisms behind right ear. Purgatives.

PARALYSIS AGITANS.—Elizabeth D., æt. 60, Married. Adm. Nov. 24. Disch. Dec. 14. In H. 20 days. *Much relieved.* Dr. Garrod, vol. 1, p. 238. Subject to "fits" 2 years. Shake of right arm and leg 8 months. Loss of power in leg. P. 96, feeble.

Tincture of conium.

MERCURIAL TREMOR.—W. W., æt. 58, Water Gilder. Adm. Sept. 17. Disch. Oct. 14. In H. 27 days. *Relieved.* Dr. Johnson, vol. 17, p. 89. At trade periodically 39 years. Second attack. First 1½ years ago. Lasted 8 months. Never salivated. At work during last 4 months. Tremor commenced in hands 2 months ago, now general. Some abd. pain with occ. Vomiting and loss of appetite. Bowels confined. Slight salivation 4 days after adm.

Iodide of potassium, first with bark and bicarbonate of potass, then with sulphate of iron and quassia. Shower baths.

HYSTERICAL HEMIPLEGIA.—Eliza H., æt. 21. Adm. Dec. 1. Disch. Dec. 9. In H. 8 days. *Unrelieved.* Dr. Beale, vol. 7. p. 19. Epileptic 8 years. Sudden loss of power and sensation in left leg 14 days ago. Lumbar spine tender. Bowels confined. Has leucorrhœa. Went out of hosp.

Sesquichloride of iron. Chloric ether, and quassia.

TUBERCULAR MENINGITIS.—Fanny W., æt. 13. Adm. Oct, 30. Disch. Nov. 13. In H. 19 days. *Recovery.* Dr. Beale, vol. 6, p. 269. Variola 3 months ago. Prev. ill. 1 day. Paroxysms of severe headache, with convulsions. Pupils dilated and often insensible to light. Free from headache on 9th day.

Calomel. Cold to head. E. L. nuchæ. Sp. ammon. arom. and acetate of ammonia. Then cod liver oil and iodide of iron.

EPILEPTIC HEMIPLEGIA.—James D., Adm. July 12. Disch. July 14. In H. 2 days. *Unrelieved.* Dr. Johnson. Left hemiplegia after an epileptic fit. Arm rigid. Sensation normal.

Ol ricini.

HEMIPLEGIA.—J. G., æt. 37, Painter. Adm. Oct. 26. Disch. Nov. 18. In H. 23 days. *Recovery.* Dr. Johnson, vol. 18, p. 65. Brought in insensible by police. Left arm and leg motionless and rigid. Tongue protruded to left. Left pupil contracted; face much distorted. Sphincters palsied. Had gout. Attack

preceded for 3 weeks by loss of memory, and pain in right temple. Complete recovery.

Castor oil and turpentine enema. Croton oil and colocynth. Ice to head. Sinapisms to feet. Afterwards iron, bark, and iodide of potassium.

HEMIPLEGIA.—J. W., æt. 30, Grocer. Adm. Nov. 23. Went out Nov. 27. In H. 4 days. Dr. Beale, vol. 6, p. 346. Sudden right hemiplegia 3½ months ago. Now right arm rigid and powerless. Sensation and memory affected. Can only say "yes" and "no." Leg and face nearly well.

Bark and dilute muriatic acid.

HEMIPLEGIA.—W. L., æt. 55, Bricklayer. Adm. June 9. Disch. Aug. 8. In H. 60 days. *Unrelieved.* Dr. Johnson, vol. 15, p. 238. Six months ago palsy and rigidity of left arm, then palsy of left leg. No loss of consciousness. Now memory impaired, mouth slightly drawn to the right; left forearm and hand weak, wasted, and rigid; left leg nearly well.

Sesquichloride of iron and quassia. Cod liver oil.

HEMIPLEGIA.—Wm. B., æt. 47. Adm. June 18. *Died* on July 6. In H. 18 days. Dr. Beale, vol. 6, p. 196. Occasionally intemperate. Lately ill-fed. On morning after adm. sudden right hemiplegia. Soon afterwards speechless, but conscious. No sensation in palsied limbs (12 days). Cannot protrude tongue. Incontinence of fæces and urine, the latter albuminous. Tongue afterwards dry and brown. No improvement. Became exhausted. P.M.—Old clot in left hemisphere, and outer halves of corpus striatum and optic thalamus. Cyst in left corpus st. Outer part of right corpus st. softened. Arteries at base healthy. Kidneys wasted.

Calomel purge. Sal volatile and liq. ammoniæ acetatis.

HEMIPLEGIA.—Edwd. L., æt. 67. Adm. July 3. Disch. Aug. 11. In H. 39 days. *Unrelieved.* Dr. Beale, vol. 6, p. 216. Five weeks ago sudden left hemiplegia, some loss of consciousness. On day before adm. palsy of face and complete loss of speech. Now slight palsy of left arm and leg. Mouth much drawn to the left; tongue protruded to right; radials rigid. 20 days later semicomatose. Sensation dull. Motions and urine passed under him. Became fatuous.

Liquor ammoniæ acetatis. Sp. ammon. arom. and chloric ether. Purgatives. Iodide of potassium.

PARAPLEGIA.—W. A., 36, Labourer. Adm. Sept. 7. Disch. Oct. 2. In H. 26 days. *Relieved.* Dr. Johnson, vol. 19, p. 51. Had a strain 14 months ago. Then pain across epigastrium. Weakness of arms 4 months later. Gradual paraplegia 7 weeks. Angular curvature of spine. Pain in left flank. Legs clumsy and numb. Sensation diminished. Dulness and crepn. over back of left lung.

Bark and iodide of potassium. Belladonna ointment to spine.

PARAPLEGIA.—W. B., æt. 21, Draughtsman. Adm. Oct. 26. Disch. Nov. 18. In H. 23 days. *Unrelieved.* Dr. Johnson, vol. 18, p. 65. Intemperate. Loss of power in legs (partial) 2½ years, with occasional slight improvement. Weakness of right hand, 3 months. Pupils irregular. Muscles plump. No syphilis. Attributed to masturbation. No improvement.

Strychnia and bark. Strychnia and iron. Shower baths. Galvanism. Purgatives.

CHRONIC ECZEMA.—C. G., æt. 4. Adm. April 15. Disch. May 23. In H. 38 days. *Much relieved.* Dr. Beale. Was discharged from K.C.H. on Feb. 28, almost well, On adm. eczematous crusts over face, neck, scalp, and arms.

Nitrate of bismuth ointment locally. Syrup of iodide of iron. Rhubarb and grey powder.

CHRONIC ECZEMA.—A. D., æt. 18, Dressmaker. Adm. Mar. 12. Disch. May

16. In H. 65 days. *Recovery.* Dr. Garrod, vol. 2, p. 76. Strumous. Two months ago vesicular eruption behind right ear, gradually spreading over scalp, back of neck, forehead, and cheeks. Quite well on 53rd day after adm.

Cod liver oil. Sulphate of iron. Liq. potassæ. Arsenitis. Locally starch and oxide of zinc.

Lepra.—E. M., æt. 55, Cook. Adm. March 19. Disch. April 4. In H. 16 days. *Much relieved.* Dr. Johnson, vol. 16, p. 149. Never quite free from it since commencement, 25 years ago. Worse 5 weeks. Eruption general.

Fowler's solution. Glycerine lotion. Hot air and hot water baths.

Psoriasis.—A. S., æt. 20, Housemaid. Adm. April 10, Disch. May 2. In H. 22 days. *Recovery.* Dr. Beale, vol. 6, p. 125. Ill. 1 month. Scaly eruption on right forearm, gradually spreading to face and left arm. Sore throat.

Fowler's solution (3 days). Then liq. ammon. acetatis. Tar lotion. Purgatives.

Erythema Nodosum?—M. S., æt. 22, Servant. Adm. June 3. Disch. June 17. In H. 14 days. *Recovery.* Dr. Garrod, vol. 1, p. 128. Prev. ill. 6 days. Pain in left ankle (1 day). Then pain and tenderness in right ankle on adm., extending over right ankle and lower ⅔rds of tibia. Pallid. Venous murmur in neck.

Iodide of potassium (3 days). Then quinine and sulphate of iron.

Tænia.—Edith P., æt. 5. Adm. May 5. Disch. May. 8. In H. 3 days. *Recovery.* Dr. Beale. First dose vomited. Headless worm voided after second dose.

Oil of male fern ʒii., in two doses.

Impetigo.—Margaret W., æt. 28. Adm. Oct. 15. Disch. Oct. 28. In H. 3 days. *Recovery.* Dr. Beale, vol. 6, p. 316. Prev. ill. 3 days. Eruption on arms, shoulders, and knees.

Castor oil. Water dressing.

Acute Pemphigus.—John M., æt. 9. Adm. Oct. 26. Disch. Nov. 21. In H. 26 days. *Recovery.* Dr. Beale, vol. 6, p. 318. Prev. ill. 11 days. On hands, elbows, knees, left eyebrow, and feet. Tongue furred.

Cod liver oil. Bark and muriatic acid. Water dressing. Benzoate of zinc ointment tried.

Erythema Nodosum.—M. A. H., æt. 30, Laundress. Adm. Oct. 22. Disch. Nov. 18. In H. 27 days. *Recovery.* Dr. Garrod, vol. 3, p. 107. Prev. ill. 6 days. Painful purple swellings over tibiæ, preceded by aching. Abscess in left groin afterwards.

Sulphate of iron and sulphuric acid. Iodide of potassium. Quinine and sulphuric acid.

Secondary Syphilis.—Renal Disease.—E. H., æt. 27, Needlewoman. Adm. April 24. Disch. May 29. In H. 35 days. *Recovery.* Dr. Garrod, vol. 1, p. 94. Syphilis 8 years ago, followed by secondaries. Pain and swelling of left ankle, right temporo-maxillary joint, and right ankle successively during last 3 months. Now left ankle pink, swelled, and painful; albumen and granular casts in urine. Amenorrhœa (12 months).

Iron and iodide of potassium. Morphia.

Secondary Syphilis.—W. B., æt. 34, Commissionaire. Adm. May 16. Disch.

May 27. In H. 11 days. *Recovery.* Dr. Garrod, vol. 2, p. 126. Slight tonsillar ulceration, 14 days. Node on sternum 9 days. Well in 10 days.
Iron guaiacum and iodide of potassium.

Secondary Syphilis.—A. B., æt. 24, Needlewoman. Adm. March 13. Disch. April 18. In H. 36 days. *Relieved.* Dr. Johnson, vol. 15, p. 137. Miscarriage 18 months ago. Scaly eruption 18 months. Sore throat 12 months. Pains in bones and larynx. Hoarseness.
Iodide of potassium (28 *days*). *Then iodide of mercury.*

Anœmia.—Ann H., æt. 18, Servant. Adm. April 17. Disch, May 16. In H. 29 days. *Recovery.* Dr. Johnson, vol. 16, p. 173. Ill 3 months. Extremely pallid. Catamenia absent. Well marked basic systolic bruit and jugular hum.
Sulphate of iron. Mixture.

Cancer of Mediastinum.—T. T., æt. 27. Plumber. Adm. March. 20. Disch. May 5. In H. 46 days. *Unrelieved.* Dr. Johnson, vol. 15, p. 145. Ill 4 months. Pains in chest and shoulders. Hæmoptysis. Enlarged superficial thoracic and cervical veins. Sudden aphonia and difficulty of breathing. Slight dysphagia. Progressive wasting. Face bloated. Glandular enlargement above right clavicle. On adm. dulness, and no breathing from left clavicle to lower border of 4th rib; same over whole left upper lobe by 20th day: and over whole of left lung (except extreme base) by 46th day; vocal cords motionless.
Quinine. Aromatic spirits of ammonia. Chlorodyne.

Anæmia.—Mary B., æt. 24. Adm. Dec. 19. Disch. Dec. 26. In H. 7 days. *Relieved.* Dr. Beale.
Calumba and sesquichloride of iron.

Anæmia.—Sarah R., æt. 28. Adm. July 18. Disch. July 28. In H. 10 days. *Relieved.* Dr. Johnson.
Sesquichloride of iron and quinine. Iron and gentian.

Purpura.—Joseph T. R., æt. 3½. Adm. July 26. Disch. Aug. 12. In H. 17 days. *Recovery.* Dr. Garrod, vol. 2, p. 242. Two days ago numerous purple spots appeared on body and limbs. Gums not spongy. No blood in evacuations. Epistaxis 3 days later.
Sulphate of iron and dilute sulphuric acid.

Purpura.—Hæmaturia.—J. A., æt. 24, Clerk. Adm. June 30. Disch. Aug. 13. In H. 44 days, *Nearly well.* Dr. Garrod, vol. 2, p. 214. Prev. ill. 5 days. Purpuric spots on legs, blood in urine, much thirst. Legs œdematous. Urine ⅓rd albumen, contains small granular casts, blood, and a little renal epithelium. Subacute rheumatism about July 10. On disch. some pain in right foot and hand—urine ⅛ albumen.
Sesquichloride of iron and quinine. Bicarbonate of potass and iodide of potassium. Then quinine and dilute sulphuric acid.

Anæmia.—Purpura.—Harriet T., æt. 19, Bootbinder. Adm. June 18. *Died* on July 12. In H. 24 days. Dr. Garrod, vol. 1, p. 145. Weakness and loss of appetite 4 months. Headache and giddiness 6 weeks. Blood in expectoration 3 weeks ago. Vomiting 1 week. Very pallid. Gums spongy and bleeding. Purple spots on arms, breath fœtid, blood in urine, basic systolic, bruit over heart, and venous murmur in neck. Eight days later, P. 120, R. 36, loud to and fro sound over heart, remaining till death. Pleuritic rub afterwards heard over left lung. Epistaxis, delirium, death from exhaustion. P.M.—Fluid in pleuræ. Recent lymph

on lower lobes of lungs. Thick warty recent lymph on heart and pericardium. But little blood in veins.—clots everywhere pale and friable.

Sulphate of iron and sulphuric acid. Citrate of iron and carbonate of ammonia. Ammonia and chloric ether. Dilute nitric acid and quassia. Brandy to 18 *oz. Chlorine gargle.*

Cancer of Rectum and Liver.—T. L., æt. 63, Porter. Adm. Dec. 13. *Died* on Dec. 18. In H. 5 days. Dr. Garrod, vol. 1, p. 259. Received from surgical ward. Winter cough 20 years. Ascites 2 months. Feet and ankles œdematous. Soft basic systolic bruit. P. 90 irregular. Bowels relaxed. Urine not albuminous. Became comatose. P.M.—Much fluid in peritoneum. Many small cancerous tumours in liver—surface of liver nodulated. Small fibrinous warts on aortic valves. Aorta atheromatous. Granular lymph on small intestines. Epithelonia of rectum. Kidneys granular.

Bitartrate of potass and jalap. Ipecacuanha wine and mist. camph. Benzoate of ammonia. Gin 6 *oz.*

Cancer of Omentum and Mesenteric Glands.—Mary R., æt. 33. Adm. Nov. 18. Disch. Dec. 2. In H. 14 days. *Unrelieved.* Dr. Johnson, vol, 19, p. 159. Loss of flesh, constant dull pain at umbilicus, and occasional vomiting (6 years). Acute pain in abdomen 8 days ago. Much emaciated. Aspect cachectic. Abdomen enlarged. Several tumours felt through its walls, apparently in omentum ; first noticed 8 days ago. Much vomiting. Urine albuminous. Feet œdematous. Discharged at her own request.

Dilute hydrocyanic acid (6 *days*). *Then iron and quinine. Opiate poultice to abdomen.*

Ovarian Cancer.—Ascites.—Dropsy of Right Pleura.—S.P., æt. 40, Married. Adm. Nov. 9. *Died* on Nov. 11. In H. 2 days. Dr. Johnson, vol. 17, p. 147. Legs œdematous 2 years. Ascites 7 months. Belly tapped one month ago, but now considerably distended. Breathing much distressed. Can only lie on right side. Bronchial breathing at base of right lung behind. P.M.—Nearly a gallon of fluid in right pleura. Much ascites. Cirrhosis of liver. Colloid cancer and cysts of right ovary—weight 4 lbs. Cancer of lumbar glands.

Aromatic spts. of ammonia, chloric ether and squills. Elaterium. Brandy.

Ovarian Tumour.—Mary P., æt. 38, Married. Adm. May 26. Transferred to surgical ward. Dr. Johnson, vol. 16, p. 221. Commenced 5 years ago on right side, after a fall down stairs. Painful at first. Tapped 9 months ago, and again three weeks afterwards. Tumour now hard, and at times painful, with fistulous opening to left of umbilicus, discharging glairy pus. Loss of flesh and strength and occasional vomiting during last 7 months.

Ovaritis.—A. J., æt. 34, Married. Adm. Dec. 7. Disch. Dec. 23. In H. 16 days. *Much relieved.* Dr. Johnson, vol. 17, p. 179. Catamenia irregular 6 months. Prev. ill. 16 days. Abdominal pain and tenderness, especially in left flank. Tympanites. Vomiting. Diarrhœa. Bearing down pains. Difficult micturition. Tenderness over lower dorsal vertebræ. Has cancer of cervix uteri.

Sal volatile and dilute hydrocyanic acid. Morphia. Castor oil. Opiate poultice to abdomen.

Uterine Tumour.—A. W., æt. 36, Married. Adm. July 29. Disch. Aug. 28. In H. 30 days. *Unrelieved.* Dr. Garrod, vol. 1, p. 193. Had 3 miscarriages, the last 2 years ago. Pain and enlargement of abdomen 1 year. Loss of flesh. Mucous discharge from vagina. Dull, hard inelastic tumour occupying the

greater part of abdomen, and a small tender tumour in right groin. Resisting mass felt in cavity of uterus by sounding. Dulness and crepitation at apex of left lung. Appetite bad.

Uterine Hæmorrhage.—H. H., æt. 40, Dressmaker. Adm. Nov. 19. Disch. Dec. 29. In H. 38 days. *Relieved.* Dr. Garrod, vol. 3, p. 155. Tumour of uterus, causing frequent hæmorrhage and difficult micturition and defecation. Much spinal tenderness. Chronic bronchitis. Transferred to K. C. ward.

Gallic acid. Iodide and bromide of potassium. Asafœtida. Ergot of rye. Belladonna plaster to back.

Leucorrhœa.—D. W., æt. 20, Married. Adm. Dec. 21. Disch. Dec. 30. In H. 9 days. *Unrelieved.* Dr. Johnson, vol. 19, p. 194. Much pain at catamenial periods. Leucorrhœa 3 years. Worse 3 months. Emaciation. Headache. Loss of appetite. Tenderness of lower dorsal spine. Diarrhœa 4 days.

Iron and quinine. Inject. Communis.

Diarrhœa.—Phlegmasia Dolens.—M. A., æt. 35, Hosieress. Adm. June 20. Disch. July 11. In H. 21 days. *Very much relieved.* Dr. Garrod, vol. 2. p. 187. Diarrhœa since 2nd day after her sixth confinement, 4 weeks ago. Three days ago legs below knees became œdematous, painful, and very tender. Very anœmic. Basic systolic bruit. Purged 3 or 4 times daily. Some epigastric pain and tenderness. Tongue white. Appetite bad.

Chloric ether, bismuth, and calumba. Logwood, dilute sulphuric acid, and calumba.

Tape-worm.—D. H., æt. 21, Clerk. Adm. Nov. 5. Disch. Nov. 11. In H. 6 days. *Recovery.* Dr. Garrod, vol. 3, p 138. Has passed joints for 9 months. Itching of anus. Headless worm came away after one dose (ʒii.) of kamala.

Kamala. Castor oil. Afterwards sulphate of iron and dilute sulphuric acid.

Tape-worm.—S. D., æt. 30, Housemaid. Adm. July 3. Disch. July 15. In H. 12 days. *Recovery.* Dr. Garrod, vol. 1, p. 159. Catamenia irregular, 4 months. Has passed joints for 3 months. Loss of appetite. Bowels generally relaxed. Headless worm came away after one dose (ʒij) of kamala.

Kamala. Castor oil.

Exhaustion.—Henry S., æt. 57. Adm. Aug. 7. Disch. Aug. 8. In H. 1 day. *Recovery.* Dr. Johnson.

Hypochondriasis.—J. C., æt. 28, Brickmaker. Adm. July 28. Disch. Aug. 15. In H. 18 days. *Relieved.* Dr. Garrod, vol. 2, p. 246. Had ague 12 years ago. Now ill 1 year. Emaciation. Loss of appetite. Tongue coated. Sleeps badly.

Quinine and dilute sulphuric acid. Asafœtida.

Debility.—Charles S., æt., æt. 12. Adm. Sept. 17. Disch. Sept. 21. In H. 4 days. *Recovery.* Dr. Garrod, vol. 3, p. 20. Typhus 4 months ago. Subacute rheum. 3 weeks ago.

Sulphate of iron, dilute sulphuric acid, and quassia.

Debility.—G. J., æt. 33, Needlewoman. Adm. March 31. Disch. May 29. In H. 59 days. *Recovery.* Dr. Johnson, vol. 15, p. 149. Hardworked. Catamenia scanty. Signs of slight tubercular deposit at right apex, disappearing while in hospital.

Quinine. Iron.

Debility.—J. S., æt. 28, Servant. Adm. May 22. Disch. June 17. In H. 26 days. *Much relieved.* Dr. Beale, vol. 5, p. 170. Hard worked. Catamenia irregular.

Iron and chloric ether.

Debility.—H. H., æt. 43. House Painter. Adm. June 11. Disch. June 20. In H. 9 days. *Recovery.* Dr. Johnson, vol. 17, p. 57. Fourteen days ago acute bronchitis. Delirious 7 days. Now convalescent, but weak.

Iron and quassia.

Debility.—D. G., æt. 63, Coach Painter. Adm. May 7. Disch. May 23. In H. 16 days. *Relieved.* Dr. Johnson, vol. 17, p. 6. Feeble. Pains in legs and back.

Iron and chloric ether. Iron and iodide of potassium.

Debility.—C. J., æt. 40, Laundress. Adm. May 23. Disch. June 20. In H. 28 days. *Relieved.* Dr. Johnson, vol. 15, p. 205. Overworked. Leucorrhœa.

Iron and quinine.

Alcoholic Delirium.—Ann P., æt. 59. Adm. March 30. Disch. April 4. In H. 5 days. *Recovery.* Dr. Johnson.

Stomach pump. Purgatives.

Dropsy.—J. S., æt. 19, Servant. Adm. Oct. 23. Disch. Oct. 31. In H. 8 days. *Recovery.* Dr. Garrod, vol. 3, p. 110. Prev. ill. 4 days. Pain in abdomen. Slight œdema of ankles. Urine not albuminous. No cardiac disease.

Bicarbonate of potass. Castor oil.

* Lead Colic and Palsy.—W. J., æt. 34, Cook. Adm. Feb. 12. Disch. May 23. In H. 100 days. *Improved.* Dr. Garrod, vol. 2, p. 42. Has for some time past taken a morning draught of beer, stagnant in leaden pipes during the night. Eight weeks ago, loss of appetite, vomiting, purging, and slight umbilical pain (1 month). Severe colic 29 days ago; remission followed by sharp attack 1 day before adm. Complete loss of power in arms, 22 days. On adm. very pallid, left hand and arm powerless, right arm feeble; brachial extensors wasted. Marked blue line on gums; blood serum loaded with uric acid. Gout in left great toe on 35th day after adm. (5 days). Gums clean on 46th day after adm. General health much improved. Slight increase of power in arms.

Treatment much varied. Chiefly iodide of potassium, sulphate of iron, strychnia, and quinine. Galvanism.

Lead Colic.—W. C., æt. 30, House Painter. Adm. July 16. Disch. Aug. 5. In H. 20 days. *Recovery.* Dr. Garrod, vol. 2, p. 196. In K. C. H. with lead colic about 3 weeks ago. Has only done 3 days work since discharge. Prev. ill. 1 day. Much abdominal pain and tenderness. Vomiting. Distinct blue line on gums. Tongue furred. Appetite bad.

Opium and belladonna. Castor oil. Iodide of potassium.

Lead Colic.—Ann M., æt. 24. Adm. Oct. 2. Disch. Oct. 14. In H. 12 days. *Recovery.* Dr. Johnson, vol. 18, p. 23. Employed last 3 weeks in white lead factory, at packing white lead. Colic 1 week, preceded by loss of appetite and occasional vomiting. Constipation 4 days. Distinct blue line on gums.

Castor oil and laudanum. Iodide of potassium and bark.

*Lead Colic.—W. C., æt. 30, House Painter. Adm. June 12. Disch. June 27. In H. 15 days. *Recovery.* Dr. Garrod, vol. 2, p. 166. Colic 6 years ago, five attacks since. Wrist drop 11 months ago. Cured in 4 months. Now ill 7 days, with nausea, slight constipation, and twisting umbilical pain. Marked blue line on gums. Urine albuminous. Quite well on 19th day.

Tr. Opii. mxx. Salts and belladonna. Hirud. vj. epigast.

Lead Poisoning.—P. McD., æt. 20, Bricklayer. Adm. June 18. Disch. June 20. In H. 2 days. *Relieved.* Dr. Garrod, vol. 1, p. 149. Bricklaying at lead works during last 3 months. Bowels regular. Ill 13 days with shivering, thirst, and headache. Slight blue line on gums. P. 120.

Liq. ammon. acetatis. ℥*p. t. d.*

Opium Poisoning.—Sarah L., æt. 50. Adm. April 10. Disch. April 11. In H. 1 day. *Recovery.* Dr. Beale. Took ℥ss, (?) of laudanum while drunk, and two hours afterwards a second ℥ss. Found almost insensible soon afterwards. When brought in, quite comatose, surface cool, breathing slow, pupils microscopic.

Stomach pump. Emetics. Strong coffee. Wet towelling. Ambulation.

Belladonna Poisoning.—Mary H., æt. 38. Adm. May 8. Disch. May 13. In H. 5 days. *Recovery.* Dr. Johnson, vol. 17, p. 19. Accidental. Dose doubtful. Symptoms an hour afterwards, lasting two days. Dryness of mouth and fauces. Burning in abdomen. Wide insensible pupils. Presbyopia. Aural noises. Deafness. Mild delirium. Weakened motor power.

Ipecacuanha emetics, stomach pump, animal charcoal, castor oil and brandy. Afterwards quinine and sesquichloride of iron.

ON A CASE OF PARAPLEGIA.

By J. Lockhart Clarke, F.R.S., &c.

History.—Mr. C., ætat 57, builder, had been a stout, powerful, active, and healthy-looking man, with black hair, dark eyes, and ruddy complexion. For some years, however, he had experienced occasional pains in the loins, causing a little difficulty in rising from his seat, or coming on while bending his body in different directions. He had also, for years, been subject to "rheumatic pains in the arms, bladebones, and knees," sometimes causing more or less lameness for several weeks together; but in the intervals between these attacks, he was perfectly well, and underwent daily a great deal of bodily exertion. During the last fourteen years he lost occasionally a considerable quantity of blood with the evacuations; and for the last five years this loss, in smaller quantities, occurred almost daily. Although a steady, industrious man, and not at all addicted to intemperance, he was yet in the habit of taking daily, by frequent draughts, a considerable quantity of old ale. His father was subject to rheumatism, and when a young man, had an attack of hemiplegia; but it is uncertain of what disease he died.

About the end of August, 1862, he began to feel a dull but c o n s t a n t aching pain over the region of the left kidney, aggravated by stooping or bending about, but not by pressure. Fourteen days later a similar pain was experienced at the corresponding part of the opposite side, accompanied by pain in front, just below the last rib, and sometimes around the navel. The latter symptom, which was often described as very severe, he regarded as "a griping of the bowels," and endeavoured to relieve by the use of stimulants and carminatives. Sometimes the pain was felt chiefly, or only, in the back; sometimes chiefly in front, and sometimes in both places, together with a sensation as if a cord was tightly tied around the waist. In the arms also, he experienced occasionally a certain degree of pain. The urine from the first was exceedingly turbid, and dark red.

Such was the information which I gleaned from the patient and his wife, when I first saw him on the 23rd December, 1862, about f o u r m o n t h s from the commencement of his illness. Then I found him almost unable, from severe pain in the back, to rise from the easy chair in which he was sitting. His face

was rather pallid, and his flesh was flabby. His urine was small in quantity, loaded with urates, but free from albumen. Yet he did not complain of feeling ill. He had no headache; his appetite was very good; his tongue was clean; his bowels acted very regularly; his pulse was quiet and moderately strong. Up to this period he had been treated by three medical men for rheumatism and lumbago.

I expressed my opinion that the patient was suffering from some affection of the membranes of the spinal cord; that the affection had probably a rheumatic origin; and that what we should have to fear would be disorganization of the cord itself, and consequent paralysis of the lower extremities. He was ordered

Liq. Potassæ, ♏ xx,
Træ. Hyoscyam, m x,
Potas. Iodidi, gr. v,

in infusion of buchu, three times a-day; half a grain of acetate of morphia every night; and a plaister of belladonna, opium, and camphor to the back. Under this treatment, in about three weeks, the urine became, for the first time since his illness, perfectly clear, and more abundant. The pain in the back was very much relieved, and, indeed, sometimes entirely absent. Now and then, for a day, the urine became thick, with increase of pain either in the back or in front; but generally, the patient had improved so much, that he was not only able to move about and stoop, but on two occasions he walked to the upper part of the house, and amused himself by doing some joiners' work. Nevertheless, he began to complain of a sensation of "pins and needles" in his feet, and of slight weakness of his legs. Some ointment containing antim. tart. was ordered to be rubbed into the back, and cod-liver oil to be taken.

About the middle of the ensuing February, during one of the occasional returns of pain with turbid urine, he was persuaded, without my knowledge, to consult a certain London physician, who regarded his case as one of rheumatism in the back, and prescribed for him a pill containing calomel and acet. colchici, to be taken for three successive nights, and to be followed each morning by an active black draught. The effect of this treatment was great prostration and a return of all the severer symptoms. He was no longer able to walk; he looked wretchedly ill and haggard; his urine became exceedingly thick and loaded with urates; and at the end of about a week (Feb. 23), being unable to stand, and scarcely able to feel his legs, he took to his bed. Now he became subject to frequent

and profuse sweating, accompanied by a small and rapid pulse. He was ordered decoct. of bark, nitric acid and opium.

From the time that he first complained of weakness of his legs, the left leg was weaker than the right; and although sensation in both was impaired, it was more so in the left. Very shortly after taking to his bed, that is about the beginning of March, he lost all voluntary power over the left leg, the toes being the parts that were last paralysed. A few days later the right leg became completely paralysed, but retained some sensibility. Both legs were very susceptible of reflex excitement. Shortly before all voluntary control over the right leg was lost, reflex action, on tickling the soles of both feet, was most marked in the left, which was completely paralysed.

About the same time, he began to experience some inability to strain, both at stool and in passing water, with a difficulty in maintaining the erect posture in bed. In the right arm, also, he now complained of slight weakness, with pains in both arms, particularly along their inner sides. The weakness gradually increased, and was soon accompanied by pains across the insides of the second row of phalanges of both hands, but particularly of the right. Great heat was also occasionally felt in their palms. While these symptoms were making their appearance, the reflex susceptibility of the cord was very strikingly manifested. The legs started whenever they were touched either by himself or by others; and whenever he extended his arms, even only to draw up the bed-clothes, the muscles of his legs were thrown into contraction. Both these phenomena, however, gradually diminished, and soon disappeared.

About the middle of March the feet and legs began to be œdematous, and there was evident effusion into the abdomen, which, like the legs, continued to increase in size.

At the beginning of April, by his own particular desire, a Smee's battery was applied once to the right arm, with the effect of removing the pain and nearly all the weakness for about half an hour. At this period all voluntary power over the abdominal muscles and sphincters was lost; the passage of the fæces and urine, however, was still felt, but only for about a week later, when the bowels became most obstinately constipated for many days together.

Hitherto he had not complained of any difficulty of breathing, but on the 8th of April, he suddenly experienced some dyspnœa and a troublesome cough, without expectoration. Two days after, a great quantity of mucus accumulated in the lungs, but

was very readily expectorated; and during the same period he complained of acute pain extending from the right hypochondriac region up to the clavicle, and to the side of the neck, with a similar pain in the neck on the opposite side.

The weakness of the right arm did not continue to increase, nor was sensation in either arm at all impaired. But in the left foot and leg, which were the first to be paralysed, all sensibility was lost by the 12th April. Yet, on pricking the soles of the foot near the heel, some reflex movements of the toes were observed. The right foot, which became quite paralysed as to voluntary motion shortly after the left, retained common sensibility, but only in a slight degree. Rubbing the instep with the hand, however, roughly, was felt only as the slightest brush. Indenting the same part with the end of the finger and the nail, was felt only as the slightest contact or touch; and pricking, or pressing on, the skin with the sharp point of an instrument was felt simply as a weight or pressure. Although, however, the left leg and foot were wholly deprived of common sensibility, they were susceptible of impressions of another kind. When a spoon, dipped in boiling water, was applied with its convex surface to the instep, a slight sensation of heat was experienced, but only after the lapse of about twelve or fourteen seconds, although the application raised vesications. In no other part of the foot, however, was the heat felt. When the hot spoon was applied to the instep of the right foot, which retained some sensibility on mechanical irritation, it felt simply warm, yet only after the lapse of about ten or twelve seconds. But when the spoon was applied to the sole of the same foot, the sensation of heat was painful, and was felt sooner after the application. With regard to the left foot there seemed to be some uncertainty in the patient's mind as to the locality of the impression, when he did not see it made; but after a little attention and consideration, he was generally correct in his answers.

Soon after these observations were made, sensation was entirely lost in every part from the waist downward. About the same time he became subject to severe rigors, alternating with profuse sweats, and recurring several times in the course of the day. The rigors lasted frequently twenty minutes, and were so severe that they shook the bed. Now also he began to complain of considerable pain, increased on pressure and percussion, along the spine, a little above the middle of the dorsal region. The bowels, which for some time past had been

obstinately constipated, now acted of themselves, and for several days continued to pour out a most astonishing quantity of semifluid and horribly-offensive fæcal matter. These evacuations had the effect of considerably reducing the size of the abdomen, which was very large and tense. The feet and legs continued to increase in size. The urine, which constantly flowed, became very offensive, and rapidly underwent decomposition. A sloughing wound which had been forming for several weeks on the sacrum increased to an enormous size, both in breadth and depth. Fits of extreme depression, with difficult respiration, recurred more frequently from day to day, and on the 6th June, 1863, the patient quietly sank, without any disturbance of the mind.

Post-mortem Appearances.—The thorax and abdomen were not opened, but I was permitted to examine the spinal cord.

In the vertebral canal, the membranes of the cord contained a large quantity of fluid,—so large a quantity that it fluctuated through its entire length, when agitated at one end. On removing them with the cord, the anterior surface of the dura-mater was found to be attached to the posterior surface of the body of the eighth dorsal vertebra,—that is, to the anterior wall of the vertebral canal at that point,—by means of a brownish, sponge-like, cancerous mass, which emitted a horribly-offensive smell. When the body of the vertebra was removed for examination, its posterior surface, to the depth of about a quarter of an inch, was seen to be destroyed by ulceration, and covered by a quantity of pus.

The dark sponge-like mass was about the third of an inch in thickness, of a somewhat conical shape, and by its base was firmly attached to the anterior surface of the dura-mater, along a length of about an inch and a-half, and a breadth of about half an inch. On the side nearer the bone, it was loose, shaggy, or villous in structure, and paler. Fragments examined under the microscope were found to consist of long cylindrical columns, more or less interwoven with each other as represented under a low power, at *a*, Fig. 1, Plate XVII, and composed of large nucleated cells, with fibres, granules, fat-globules, and some free nuclei. Fig. 2 represents the appearance of the isolated cells under a power of 420 diameters. These were extremely delicate, and very finely granular. In the majority of instances each contained a large and conspicuous nucleus, with one nucleolus or more. Sometimes the nucleus was double, but occasionally it was entirely absent, and replaced by numerous fat globules, which were either scattered through the cell or collected together at one part. Every cell gave origin to

two processes or more, which, in many instances, were observed to have dilatations along their course, as at *b*, Fig. 2. The shape of the cells was very diversified, being round, oval, or pyriform, crescentic, triangular, or irregular, and such, that in their undisturbed positions they fitted each other in close apposition, or dove-tailed exactly, to form the beautifully-compact and somewhat cylindrical columns to which the shaggy or villous appearance of the mass was due (see Fig. 3). These columns were separated from each other, more or less widely and completely, by bundles of nucleated fibres, with which the processes of their cells were continuous (Fig. 3).

On proceeding from this loose or superficial portion of the mass towards the surface of the dura-mater, the columns of cells assumed first an oblique, and then a more or less horizontal direction, as shown at *d e*, Fig. 1, which represents a small piece of a longitudinal section, magnified about 35 diameters. In the same proportion they became more and more closely interlaced; and in the deepest strata, as at *e*, they were frequently twisted or contorted in different directions into loops of various lengths, or they enclosed spaces of a pyriform, oval, or circular form. A piece showing these loops and contortions, magnified about 100 diameters, is represented in Fig. 7; and another piece exhibiting two of the holes or vacant spaces, and magnified 220 diameters, is represented in Fig. 8. The arrangement of the cells at the circumference of these spaces very commonly resembled that around the cavities of glandular structures, especially of the subepithelial glandular layer of the olfactory mucous membrane. Between these cavities and loops, nucleated fibres, collected into bundles of various sizes (*m*, *m*, *m*), ran in different directions, forming a kind of plexus, and uniting with the processes of the cells. At *n*, many of the cells which covered one of these bundles, and belonged to the group *o*, through which it passed, have been torn from its surface, exposing the fibres and nuclei. Between the groups of cells densely aggregated groups of small nuclei were frequently observed, and amongst these, or amongst large bundles of fibrous tissue in their vicinity, a variable number of pigment granules or grains were either scattered or collected into clusters; but no pigment was ever discovered amongst the large cells and the fibres composing the loops and villous processes already described and represented in Figs. 3, 7, and 8.

In some parts of the cancerous mass, this kind of structure sprang directly from the surface of the dura-mater (*f*, Fig. 1), with the fibres of which its own were directly continuous; but in most places it was connected with the dura-mater through

the medium of another layer of a somewhat different tissue, and of considerable but variable depth, intervening between *e* and *f*, Fig. 1. This structure was much more compact than the one just described, of a darker brown, and bore at different intervals two or three large, and many small, black, or melanoid patches. Generally it consisted of an extremely delicate network or matrix of the finest connective tissue, holding together nucleated fibres, nucleated cells, pigment-particles, and free nuclei.

The nucleated fibres had in some places a nearly horizontal direction: frequently their course was perpendicular or oblique, or in every direction between the other elementary bodies, which were scattered about without regularity or order. The nucleated cells, in a few spots, were similar to those of the loops and processes in the villous or superjacent layer; but the majority, by far, were of smaller average size, and of a different character and shape. Generally, they were more or less round, oval, or heart-shaped. Some of them, as at *h*, *i*, Fig. 4, were filled or only partially filled, with groups of smaller cells, which gave them a kind of mulberry appearance. These were occasionally tinted, in a greater or less degree, by pigment in a state of solution. The majority were somewhat smaller, as represented at *k*, Fig. 4. Many of them contained large and distinct nuclei, others only granules, with two or three small and globular bodies. Some were clear, or perfectly free from pigment; some were variously or partially tinted by this substance in solution; while others contained it in the form of granules or grains, which either accumulated on one side, or surrounded the nuclei, and obscured them wholly or in part.

The pigment, however, was also found in what is called a free state, or outside the cells (Fig. 5). But in many instances this could not with propriety be called a free state, for numbers of the particles consisted of cells or nuclei, which had been filled or choked up by pigment. Sometimes the particles or granules were completely isolated. Sometimes they formed into clusters of different sizes, and these again into groups, and in all these forms they were scattered through the layer, and collected at intervals into denser aggregations of variable shape and size. The browner tint which, as already stated, belonged to this layer, was due to the pigment dispersed through the structure; and the black or melanoid patches above mentioned were parts in which the pigmentary particles were in denser groups.

The free nuclei, *l*, Fig. 4, were for the most part round or oval, and varied from the diameter of a blood disc to about double that size. Like the cells, they were frequently tinted by pigment in solution, and scattered irregularly through the

layer; but in some places they were densely crowded together, with pigment granules, into groups of different sizes, particularly around oval or elongated spaces, and in bands of fibrous tissue near the surface of the dura-mater.

But besides the elementary bodies above described, small and variously-shaped masses of a yellowish, opaque, and hard substance were found embedded in different parts of the layer. This substance, as seen in very thin sections, consisted of a network of short and exceedingly tough fibres. In some places the fibres were extremely fine, and arranged for the most part in nearly one direction, but crossed at right angles by others; *p*, Fig. 6 represents a thin section in the average direction of the majority of these fibres, magnified 670 diameters; and *q* another section in a plane at right angles to them. On the application of acetic acid, small, delicate, smooth, and isolated nuclei made their appearance, but were sparingly scattered through the tissue. In other parts the fibres were coarser, more crooked and ragged; the meshes they enclosed were larger, and the reticulated tissue which they formed was like the cancerous structure of bone. Here the nuclei were more abundant, and sometimes collected into dense heaps. Many of them were much larger than in other places, as large as three times the diameter of a blood disc. Frequently their surface was uneven, imperfectly defined and granular.

There can be no doubt that this tough fibrous tissue, upon which hydrochloric acid had no perceptible effect, was the organic basis of osteoid cancer, in which the salts of lime had not been deposited.

But the organic basis of bone was not the only tissue of the kind that was found in this layer. Pieces of cartilage of different shapes and sizes, were embedded in bundles of fibrous tissue nearer the surface of the dura-mater.

There was one other structure of a remarkable kind found in many parts of this layer. It consisted of a peculiar substance spread out in broad layers, or contained in tubes, of which the walls and the tissue between them were composed of nucleated fibres, and nucleated cells of a round or oval form. Fig. 9 represents a transverse section of a group of such tubes, with the intervening tissue magnified 90 diameters. The contained substance consisted of round or hexagonal cells in close apposition, and about the size of blood-globules. Indeed, at first sight, I regarded these tubes as blood-vessels, surrounded by layers of cancer-cells, and their contents as blood-globules somewhat altered by the chromic acid solution in which they had been preserved. On closer observation, however, they ap-

peared to be of a different nature; for from the walls of the tubes or cavities processes extended inwards, and divided in their course into filaments, which became connected with the cellular bodies, as represented at *p*, *p*, Fig. 9, and at Fig. 10, magnified 420 diameters, where *q* is one of the processes dividing between the cells. Embedded amongst these, and also in connection with the fibres, there were other bodies of a darker and coarser description, as shown at *r*, *r*. This delicate tissue, probably, was originally gelatinous or fluid, and instead of being contained in tubes, was often spread out in broad expansions. In the vicinity, as at many other spots, groups of large fat-cells were observed, containing a variable number of acicular crystals of margaric acid.

The morbid growth, of which I have now completed the description, is an interesting example of the close relation between different forms of cancer. The looser and more superficial portion is of that kind which is known to pathologists as villous cancer, and was first well described by Rokitansky, who regards it as a form of medullary cancer with predominant stroma-formation.*

The deeper and darker layer of this malignant growth was, in most parts, allied to the harder forms of medullary cancer; in many places, to the melanoid, which may be regarded as a kind of medullary containing a large quantity of pigment; and in a few places we saw the presence of cartilage, and a tendency to the formation of bone, or of the osteoid variety of cancer.

Morbid Anatomy of the Spinal Cord.—Opposite the eighth dorsal or diseased vertebra, the cord was somewhat constricted, and morbid appearances of variable extent were discovered throughout its thickness. In many sections, portions of the grey substance, particularly in the neighbourhood of the tractus intermedio-lateralis and the anterior cornu, were partially or wholly destroyed by pale, transparent, and semifluid disintegration. In all the white columns the same kind of lesion affected small and irregular areas, which were sometimes isolated by the surrounding tissue, but more frequently they coalesced in different degrees, at different points, and in such a way as to give the appearance of a moth-eaten tissue. In these transparent tracts of disintegration, a small number of granular, so-called exudation, corpuscles were some-

* Ueber die Entwickelung der Krebsgerüste mit Hinblick auf das Wesen und die Entwickelung anderer Malchenwerke. 1862. *Sitzungsberichte der Kaiserlichen Akademie* of Vienna.

Ueber den Zettenkrebs. *Ibid.*

The first of these is on the stroma of cancer; the second on villous cancer.

times observed. The white columns, moreover, were affected by another kind of lesion, which I described on a former occasion.* This consisted of partial or entire disintegration of the white substance of many of the longitudinal nerve-fibres; and when seen in transverse sections under a low power, gave to the columns a kind of honey-comb appearance, or an appearance resembling that presented by transverse sections of the stems of certain plants.

Such, nearly, was the condition of the cord as far as the lower end of the dorsal region. Here the posterior and lateral white columns were much more healthy, but the anterior columns were softened and extensively diseased. Contrary to the normal relation, the diameter of the cord was greater than immediately below. The grey substance was very much diseased, mis-shapen, and unsymmetrical on the opposite sides (see Plate XVIII, fig. 11). Nowhere could it be considered perfectly healthy; but in the anterior and lateral parts, especially, there were several large and irregular areas of transparent disintegration, *a*, *a*, *a*, in which the tissue was very much destroyed, for it consisted only of a few fibres and blood-vessels stretched across the space, with the *débris* of the remaining elements.† A large portion of the anterior cornu of the left side, at *b*, was in a similar state of disintegration, only a few damaged fibres of the anterior roots, and a few partially-eroded blood-vessels escaping, embedded in a finely-granular and semifluid substance. Not a vestige of a nerve-cell could be discovered. At the bottom of the anterior median fissure, the triangular space *d*, which was likewise filled with a granular, semifluid substance, the blood-vessels were either wholly or partially destroyed.

A little lower down in the cord, the anterior columns were damaged to a still greater degree, and the anterior cornu on the right side was partly obliterated, while that on the left was disorganised and semifluid. At the lowest part of this region, that is, at the twelfth dorsal nerves, the whole thickness of the cord, but especially the anterior half, were so completely broken down that it was quite impossible to make any sections whatever.

Throughout the whole of the lumbar enlargement, these morbid appearances diminished considerably. With the exception of some damage to the fibres of the anterior commissure, every part of the grey substance appeared to be nearly healthy;

* British and Foreign Med.-Chirurg. Review. April 1864, p. 491, and fig. 5, B.

† This fig., as well as the next, fig. 12, has been turned by the engraver with the *anterior* side upward, which is contrary to the way in which I have been in the habit of representing sections of the spinal cord.

but in all the white columns the fibres were extensively disintegrated, the areas of disintegration, in transverse sections, appearing either as isolated points, or as irregular patches, which in various ways coalesced with each other and with the natural fissures, along the edges of which the same kind of disintegration had taken place. In all these morbid spaces, as well as in the connective tissue throughout the white columns, fat-globules of different sizes were thickly interspersed.

In the upper two-thirds of the conus medullaris, both the grey and white substances continued to be nearly in the condition just described; but at the lower third of this region, the central parts of the grey substance for some distance around the canal, were reduced to a granular, very transparent, and almost fluid state.

Starting afresh from the diseased vertebra, I shall now proceed to describe the condition of the upper parts of the cord. At the seventh pair of dorsal nerves morbid appearances were extensive throughout its entire thickness. All the white columns were damaged by a large number of the round or oval points of the disintegration, to which I have already alluded; and the same kind of destruction had invaded the edges of the numerous natural fissures, which thus were more or less widened, and either partially vacant or completely filled up by the *débris* of the adjacent tissue. In the grey substance, the commissures, the parts around the canal, and the extremities of both the anterior cornua, were variously affected by granular disorganization. In some sections, the two sides were quite unsymmetrical, the right posterior cornu being strangely twisted, and dragged in that direction by a hernial protrusion or nodular swelling of the right lateral column. At the same level, also, the deep portions of the posterior white columns between the cornua were very soft, and for a few sections upward, were quite pulpy.

At the sixth dorsal nerves, the cord, for about an inch in length, was dilated and very much diseased. The posterior columns were as soft as in the parts we have just left. The anterior median fissure was very much drawn to the left side from within outward, so that the right anterior column strangely overlapped the left, and appeared to be much larger. The lesions of structure were remarkable in both, but particularly in the right one; for its deeper half was replaced by several fluid or semifluid areas, which variously coalesced, and extended into the anterior cornu, where they likewise destroyed a large portion of the grey substance. The rest of this substance was much displaced and misshapen. Toward the upper end of

this dilatation of the cord, the areas of disintegration gradually diminished; the anterior median fissure in the same proportion regained the middle line; and the anterior column on the right side resumed its normal size; but the whole interior of the cord was softened, and the deeper portions of the posterior columns between the cornua were reduced to a complete pulp.

For about an inch above this dilatation, the diameter of the cord was normal; but at the fifth dorsal nerves it was again slightly dilated for the length of about half an inch. At this point the deep portions of the posterior columns between the cornua were quite pulpy, and this state of softening extended across the right cervix cornu—which it quite destroyed—into the corresponding lateral column, where the surface was raised into another nodule or hernial protrusion. On both sides all the grey substance, but particularly the anterior and central parts, were variously and extensively damaged by granular disintegration.

For a space of about half an inch above this second dilatation, the cord was not quite so soft, but at the lowest root of the fourth dorsal nerves, the lesions became more extensive; for in the grey substance the softening had resulted in the formation of numerous fluid spaces of a round, oval, or irregular shape and very variable size. In some instances the round or oval spaces were very sharply circumscribed, and contained a fluid which was either perfectly pellucid, or rendered more or less turbid by the granular *débris* of the tissue which it replaced. In other instances the lesions were most irregular, and frequently resembled a piece of cloth that has been worn into threads or stretched into numerous and adjacent slits, with intervening fibres that cross each other, or meet at different angles, like the fibres of a tangled skein. All the white columns, but especially the anterior, were more or less softened, or otherwise damaged. At the upper half of the roots of these nerves (the fourth dorsal), the peculiar patches of fluid disintegration almost wholly disappeared, although the grey substance was in many places injured or displaced, and the posterior columns between the cornua were much softened.

At the third dorsal nerves the white columns remained nearly in the same condition; but in the grey substance, especially in the vicinity of the commissures, of the anterior cornua and the base of the posterior, fresh lesions reappeared. Through the second dorsal nerves these continued to increase, and fluid spaces of various shapes, and sometimes of enormous size, destroyed portions of both the white and grey substance. Fig. 12 represents a transverse section of the cord at the middle

of the second dorsal nerves.* *e*, *e'* are the posterior white columns; *f*, *f'* the antero-lateral columns; *g*, *g'* the caput cornu posterioris, or dilated extremity of the posterior horn, *g'* being its gelatinous substance; *h*, *h'* is the cervix cornu or constricted portion of the horn; *i* is the tractus intermedio-lateralis; *j*, the anterior cornu. Now on the left side, the caput cornu posterioris, *g*, *g'* appeared to be nearly healthy; but the cervix cornu *h*, *h'* was extensively injured by pale streaks and patches of disintegration; while the tractus intermedio-lateralis, *i*, and the central parts of the lateral grey substance, as far as the base of the anterior cornu *j*, consisted of nothing but a finely granular and transparent mass, with fibres stretching quite across, or projecting from its opposite sides. The antero-lateral columns were but little injured. On the right side, however, where they are represented dark, their deeper layer of fibres, along the line *g'*, *g*, *h*, *h'*, *i*, had suffered almost complete solution, their *débris* blending with that of the grey substance in extensive areas of disintegration, as represented by the blank spaces. In the deep portion of the posterior columns *e'* was a somewhat circular space, in which the same kind of destructive process had reduced the tissue to a state which resembles a piece of cloth that has worn into irregular holes.

For a few sections higher, these structural lesions diminished to a certain extent; but the white columns, particularly those between the posterior cornua, were softer than natural.

From the middle of these roots to the middle of the first dorsal nerves, the cord was again somewhat swollen; and at the lower part of this third dilatation, the commissures were almost wholly destroyed. On the right side, nearly the whole of the grey substance, and a large portion of the lateral column, were reduced to a state of solution. For a few sections higher, the right anterior cornu was very much swollen or larger than natural, and the part around the canal contained several patches of disintegration; but the lateral columns were not so much affected, although softened to a considerable degree, particularly along the edges of the natural fissures, where the fibres were reduced to granules.

At the eighth cervical nerves the white columns were much firmer and more healthy, but the lesions in the grey substance were more extensive. In some sections the anterior and posterior commissures were partially destroyed, and in most sections a large portion of the grey substance on each side was

* In consequence of the figure having been reversed in the printing, the right side in the text corresponds to the left side in the figure.

rendered very transparent and more or less fluid by extensive patches of granular disintegration. Almost the only parts that escaped were the caput cornu posterioris, and the extremity of the anterior horn. The blood-vessels that enter at the anterior median fissure were frequently more or less destroyed; and the fissure itself was sometimes filled with a fluid or semi-fluid substance, containing the *débris* of the blood-vessels, and of the edges at the adjacent columns.

In ascending along the seventh cervical nerves, the lesions of structure gradually diminished, and at the upper part of the sixth pair they entirely disappeared. These lesions there were quite sufficient to account for the weakness and pain which the patient experienced in his upper extremities.

Of the spinal cord and cancerous mass described in this communication, I have preserved upwards of fifty preparations, including those from which the illustrations were drawn.

THE GRAY SUBSTANCE OF THE MEDULLA-OBLONGATA AND TRAPEZIUM. By John Dean, M.D. Smithsonian Contribution, Philadelphia, 1863. Folio, pp. 75, with portfolio of photographs.

By W. B. Kesteven, F.R.C.S.

In the medulla-oblongata are collected all those ganglionic centres which are in association with the nerves connected with vital organs. Its structure cannot, therefore, fail to possess the deepest interest with physiologists and pathologists, and hence the importance of a just appreciation of the researches of Mr. Lockhart Clarke, and of the author of this treatise.

The work before us is divided into two parts. In the first is contained the description of the morphological changes in the medulla of the sheep and of man; of the hypoglossal nucleus and roots; of the vagus and glossopharyngeal nuclei and roots; and of the olivary bodies, and antero-lateral nucleus. In the second part is contained the description of the trapezium and its morphological changes; of the auditory, facial, and abducens and nuclei and roots; and of the upper olivary bodies.

From the nature and contents of this work, consisting as it does almost exclusively of anatomical details, it would be to transcribe these to do full justice thereto. We shall, therefore, content ourselves with epitomizing the statements it contains with reference to some of the morphological changes which make their appearance in this, the most important of the nervous centres. In the medulla spinalis, as Mr. Clarke ob-

serves, "the columns of white and grey substance are parallel to each other, and preserve the same relative position to each other throughout their entire course; but in the medulla-oblongata these parts not only assume a different arrangement, by becoming more or less blended with each other and with new structures, but frequently pursue a curvilinear direction in different planes inclined at varying angles." "The first change in the form of the grey substance as it passes from the cord to the medulla-oblongata consists in a gradual pushing outwards and forwards of the posterior cornua, which are now traversed along the anterior edge of the caput cornu, especially at its junction with the cervix, by very numerous bundles of longitudinal fibres forming a beautiful network along the lateral border of the gray substance. At the same time the anterior cornua have rapidly diminished in size, being encroached upon by similar longitudinal fasciculi, extending the above-mentioned network into the antero-lateral and anterior columns. The network connected with the posterior cornua is traversed by the upper cervical, and higher up by the hypoglossal roots."—Dean, p. 1.

The tuberculo cinereo of Rolando appears on the outer surface. This, as pointed out by Clarke, is produced by the gelatinous substance of the posterior cornu reaching the surface, and as it ascends enlarging to form a prominent column, which, near the olivary bodies, becomes overlaid and concealed by a new system of transverse arciform fibres.

A little above the decussation of the pyramids the anterior cornua have almost entirely disappeared, while the hypoglossal nucleus has become quite distinct. Along the margin of the pyramids an elongated tract of cells may be observed, the commencement of the olivary bodies. The nucleus of the spinal accessory is plainly seen as a long tract of cells reaching from the tractus intermedio-lateralis to a point just behind the central canal.

Ascending to a slight extent, we find a change in the form of the central grey substance. The posterior fissure is reduced to a sulcus; the central canal is pushed backward, and behind it the nuclei of the spinal accessory are seen. The sulcus soon afterwards opens into the fourth ventricle, just in front of the apex, where are to be found the large multipolar cells of the hypoglossal nuclei, from which the root fibres may be seen radiating to the surface between the pyramidal and olivary columns. In the posterior part of the vagus nucleus may be traced the first appearance of the auditory ganglion. The vagus nucleus, which is placed posteriorily to the hypoglossal,

is now fully formed. The auditory nucleus, as first pointed out by Lockhart Clarke, is here developed, as well as the substance of the post-pyramidal and vagal nuclei, between which it is inserted like a wedge. In like manner, higher up, the glosso-pharyngeal nucleus is formed from an extension upwards of the vagus nucleus.

The posterior vesicular column and the tractus intermedio-lateralis are continued up into the medulla as the rectiform and post-pyramidal nuclei, and into the nucleus of the spinal accessory and vagus.

"These columns, moreover, though not always so compact and well-defined as they are to be seen in the region of the accessory and vagus, may, nevertheless, be traced continuously throughout the medulla in close connection with the spinal accessory, vagus, and glosso-pharyngeal, as well as part of the auditory and trifacial roots."

The tractus intermedio-lateralis gradually disappears, becoming merged in the nucleus of the spinal accessory nerve. The anterior cornua diminish in size while its cells become collected into two groups, forming the first indication of the great hypoglossal nuclei. A group of cells exists near the border of the arciform fibres, becoming more and more prominent until the level of the vagus is reached, when it is broken up into smaller groups. This group Dean proposes to name the antero-lateral group. It is penetrated by fibres of the arciform plexus. After the ending of the decussation of the pyramids the raphè appears, formed by the union of longitudinal with arciform fibres. The olivary bodies next make their appearance, composed of layers of cells penetrated by arciform fibres, by means of which also they are connected with each other, and with the raphè. These bodies are also in connection with the hypoglossal nuclei by bundles of fibres or by the interposition of cell nuclei.

The olivary bodies are developed among the network into which the anterior cornua are resolved; they are placed behind the pyramids, and close to the course of the hypoglossal roots, consisting of a collection of cells in convoluted laminæ, separated by thick bundles of transverse fibres, which, passing inwardly, traverse the raphè, and thus form a commissure between the olivary bodies. Groups of cells are often seen near them, sometimes assuming a convoluted outline; these are the accessory olivary nuclei. "Fibres from all the nuclei of the medulla pass among the cells of the olivary bodies, and in many cases are, doubtless, continuous with their processes, so that the probabilities deduced by Clarke, from the study of

transverse sections, viz., that the olivary bodies are co-ordinating centres for the different ganglia or nuclei of the m e d u l l a - o b l o n g a t a, derives additional strength from the study of the laminæ in the beautiful convolutions in which we find them arranged in their longitudinal course. Dr. Dean claims the credit of first describing the column of large cells, which he designates the a n t e r o - l a t e r a l nucleus; which, from the variety of its connections, would seem to be of considerable importance. We shall, in order to do justice to the author, describe this nucleus in his own words :—

"It is developed in the antero-lateral portion of the medulla, just above the decussation of the pyramids, among the great bundles of arciform fibres which are here so conspicuous. The cells vary, as stated by Clarke, both in form and size, but are mostly quite large, measuring in the sheep, from $\frac{1}{800}$ to $\frac{1}{400}$ of an inch in the principal group; while behind a smaller group of cells is often found, connecting the main nucleus with the caput cornu, some of the cells of which measure from $\frac{1}{400}$ to $\frac{1}{200}$ of an inch in diameter. The cells composing the body of the nucleus are disposed in a compact group, through which the arciform fibres radiate in various wavy bundles, forming an intricate network around the longitudinal fasciculi, which are embraced in every direction by the cell fibres. The large cells mentioned above, sometimes appear as a distinct group, just on the anterior border of the caput cornu, but are more commonly an off-shoot from the main nucleus with which they are evidently closely connected. In the cat, the cells of the antero-lateral nucleus, are very conspicuous and well-defined; they are collected into a group in the same situation as in the sheep, but form a rather more open plexus or network with the arciform fibres, many of which are seen to be continuous with their processes. As cell processes also pass in many different directions, transversely as well as longitudinally, this group doubtless serves to connect the arciform fibres with the other parts of the medulla, constituting, apparently, an accessory nucleus to each olivary body.

"Its connections with the caput cornu have already been pointed out; connections are also established between this nucleus and that of the hypoglossal, and with the vagal and spinal accessory nuclei; with the two latter sometimes directly, and sometimes through the caput. The connections between the antero-lateral nucleus and the hypoglossal are often very striking. . . . . In the human medulla the antero-lateral nucleus is quite conspicuous; the cells are rather smaller than in the sheep, measuring about $\frac{1}{1000}$ to $\frac{1}{400}$

of an inch. The relations between the antero-lateral nucleus and the other portions of the medulla are the same in man as those described above in the sheep." . . . . . The large cells, the origin of the upper olivary bodies are evidently developed from the remains of the antero-lateral nuclei, so that it is extremely probable that the antero-lateral nucleus is, in the lower part of the medulla, accessory to the olivary column, and that it is continued upwards into the trapezium where it is developed into the upper olivary body.—P. 41, 42.

In a quotation which the author makes from Mr. Lockhart Clarke's paper in the "Philosophical Transactions," 1848, (p. 246) this nucleus is described in fewer words, although it was not specially named by that observer.

One prominent feature Dr. Dean's work presents is the abundance and beauty of its illustrations. These comprise no less than sixteen plates, half of which are printed copies of photographs of thirty-six opaque sections from different parts of the medulla. The author, moreover, has published a separate portfolio of photographs of these same sections. These afford, indeed, faithful representations of what may be observed on sections viewed by reflected light; but beautiful as they are, regarded as specimens of photography, they are not comparable in their usefulness to the other half of the plates, which contain thirty illustrative and explanatory figures of structures seen by transmitted light, besides five woodcuts in connection with the text. Obviously these as far transcend in value the photographs as a transparent surpasses an opaque section in the insight it permits into the distribution of nerve fibres, or the structure of ganglia. It was by the employment of means to render transparent his sections of the spinal cord that Mr. Lockhart Clarke was enabled so rapidly and decisively to advance upon the researches of Stilling and all previous anatomists. The use of photography, therefore, other than for the preservation of the arrangements revealed in transparent sections, is pro tanto a return to the darkness of opacity. Although, therefore, these photographs do not add to, they in no degree detract from, the exactness and fulness of anatomical details that will make Dr. Dean's a work indispensable to all who would be informed on the anatomy of the medulla-oblongata.

It may be acceptable to some of our readers to know the author's mode of preparation of the medulla for microscopical purposes. This consists in a slight modification of Clarke's method. "For the general study of the course and destination of fibres, I have found specimens hardened in

chromic acid, and made transparent by Clarke's method, particularly well suited. These specimens have usually been immersed for a few weeks in a solution of chromic acid of about the strength given by Clarke, and subsequently put into alcohol, by which means the specimens are hardened without becoming so brittle as is often the case if they are kept for a long time in a solution of chromic acid or bichromate of potash. I have also continued the use of copal varnish in the place of Canada balsam, as it seems to me on many accounts more advantageous than the balsam."—P. 66.

Our own experience of the use of copal varnish does not lead us to agree with the author's estimate. We have found preparations of the cord spoilt by the contraction of the varnish in drying. It should also be observed that delicate minute nerve cells are often destroyed or much changed by the chemical action of alcohol, while the weak solution of chromic acid is not open to this objection.

If our readers are familiar with the researches of Mr. Lockhart Clarke upon the anatomy of the medulla-oblongata, they will derive much pleasure from the study of Dr. Dean's work, inasmuch as it fully confirms and amplifies all the observations of our distinguished fellow-countryman. If they should not be as familiar as we could desire, for their own sakes, with Mr. Clarke's investigations, we would refer them to the Philosophical Transactions, and to the first volumes of the "Archives," where they will find, by Mr. Clarke himself, a succinct statement of the histological elements of the spinal cord. A full account of the result of the labours of the same anatomist, together with those of other observers, may also be found in the second volume of the "Natural History Review."

NOTES OF OUT-DOOR PRACTICE AT KING'S COLLEGE HOSPITAL.

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In the following paper I have selected a few of the more interesting cases from those under my care at King's College Hospital, which seem to me to be worthy of record, either on account of their rarity and importance, or from the light they throw on mooted questions of practice.

Case I.—*Laryngismus Stridulus consequent on intestinal derangement.—Remarks.*

Caroline Gray, æt. 4 months, was brought to me on the 1st of January, 1864. She had been in good health until a week before her mother applied for advice, when it was noticed that the fingers were constantly kept flexed on the palms of the hands, and on the next day she had a violent attack of convulsions. This went off, but on the following day she had as many as eight separate attacks of well-marked laryngismus, which were so violent as to threaten immediate suffocation. For some days before her illness commenced she had suffered from diarrhœa, which was still continuing. For the last two months she had been brought up by hand. She was ordered the following mixture :—

℞
Acid. Hydrocyan. dil. m. iv.
Tr. Assafœtidæ.
Liq. Chinchon. Pallid aa ʒss.
Mist. Eth. Chloric ʒj.
ʒi. 4ta. q. h.

with ½ a grain of Dover's powder at bed-time to check the diarrhœa, and 5 minims of brandy every third hour. The mother had also directions as to the better management of the infant's diet, and was recommended to employ oleaginous inunctions night and morning. Until the 1st of February there was little improvement, the fits of laryngismus still recurring with great violence on the slightest provocation, and constantly placing the child's life in jeopardy. The thumbs were all this time firmly clenched on the palms of the hands, and the toes rigidly extended. The diarrhœa also proved very troublesome, and she was now ordered a teaspoonful of raw meat grated three times a-day, which she took with avidity and great benefit, the diarrhœa rapidly disappearing. The fits became gradually less frequent, occurring not oftener than once or twice a-day, and being at the same time less severe. The medicine was now discontinued, and cod-liver oil with syrup of iodide of iron prescribed in its place. On the 12th she was so far better that the mother was advised to take her to the country, and she remained there for six weeks without any tendency to laryngismus. On her return to town, however, the bowels soon became again disordered, and the same train of symptoms showed themselves,

commencing with flexure of the thumbs, and culminating in general convulsions with spasm of the glottis, five fits having occurred within twenty-four hours. Under a similar course of treatment she again improved, and was then sent back to the country, where she has since remained in excellent health.

Remarks.—This case illustrates remarkably well the peculiarities of the disease. Here we have a child previously in tolerable health, but in all probability of weakly constitution, and, after the bowels had been for some time deranged, the whole nervous system was thrown into such an irritable condition, that the life of the child was seriously endangered by the frequent occurrence of laryngeal spasm, accompanied by general convulsions. The starting point of the disease in this case was, undoubtedly, intestinal disorder, produced by injudicious feeding; and it may be noted that the case also proves an exception to the rule of some authors, that laryngismus always occurs during dentition. It is seldom that the attacks are so frequent and severe as they were in this instance. Indeed, they recurred so often, and with such severity, that I was in constant dread of the child's being carried off in one of them, and its recovery was in a great measure due to the mother's constant and intelligent care. In many of the severer attacks chloroform was administered with great benefit; but valuable as this remedy is, it is clearly inapplicable in the majority of cases, since it can only be administered by the medical man himself. The fits rapidly diminished in intensity and severity under the tonic and anti-spasmodic treatment adopted, and would, probably, have done so sooner had we been able to check the diarrhœa, which was the immediate originator of the complaint. And when we succeeded in doing so by the use of raw meat, an immediate improvement commenced, and the case was completed by change of air. When the precautions enjoined were relaxed, disorder of the bowels again showed itself, and this was followed by a precisely similar train of symptoms. No case could better show the importance of directly attacking the root of the disease in all cases of the kind, and it is seldom that a definite cause cannot be ascertained.

CASE II.—*Invagination of Intestine.—Treatment by Insufflation.—Death.—Autopsy.*

F. G., æt. 4 months. The little boy who was the subject of this case, had been in perfect health until Friday, the 10th of March, 1865, when he was suddenly seized with severe vomiting.

The mother administered several strong purgative doses which had no effect, and on the Saturday and Sunday the child was very ill, vomiting incessantly, and occasionally passing small quantities of pure blood per rectum. On Monday, the 13th, he was brought to the hospital, and it was noted that he lay in his mother's arms with an anxious expression of countenance, constantly vomiting, and with a distended and very tender abdomen. It was ascertained by careful examination that no hernia existed, and in the course of the descending and transverse colon there was a firm, elongated tumour, hard and sensitive to pressure. The symptoms of intestinal obstruction were very clear, and, as the mother had already tried the administration of purgatives without avail, opiates were freely exhibited. Next day there was no relief, and the treatment by insufflation was attempted, the nozzle of a small pair of bellows being passed within the anus, and the injection of air continued for some time. This was followed by some relief, the child seemed to be in less pain, and from that time the vomiting ceased, but the bowels were not moved, and pure blood was still passed at intervals. On the following day insufflation was again employed with no better results, and on the 14th the child died, that is, six days from the commencement of the symptoms.

On *post-mortem* examination the small intestines were found to be largely distended with air, and the vessels much injected. No peritonitis had existed. At the ileo-cœcal valve invagination of the ileum had taken place, and the whole of the large intestine was filled with ileum as far as the commencement of the rectum, which was empty and collapsed.

Remarks.—This case is chiefly remarkable from the great extent of the invagination, which involved the whole of the ascending and transverse colon, as well as part of the descending. It is quite clear that under such circumstances no possible treatment could have been of any service, and it is evident that insufflation, to however great an extent it might have been practised, could have had no effect in relieving the obstruction. Nor need this failure be considered as in any way militating against the employment of this remedy, which has frequently been followed by the best results. For invagination to such an extent must be of exceeding rarity, and no other plan of treatment seems to be so well adapted for unfolding any obstruction that may exist; and as the invagination in children almost invariably occupies the large intestine, it would seem to be specially applicable to them. In this case the diagnosis was particularly clear, the seat of obstruction was apparently pointed out by the tumour perceptible on palpation,

and, as a forlorn hope, after the failure of the means employed, gastrotomy was contemplated. This desperate expedient has been recommended in hopeless cases, but I do not know of any instance in which it has been practised. Probably no case, as far as appearances go, could have been better adapted for it than this, yet it is clear that it would have been worse than useless, since it would have been scarcely possible to unfold so large an invagination with the slightest prospect of success.

CASE III.—*Invagination of Intestine.—Insufflation, followed by disappearance of the symptoms.—Recurrence.—Death.—Autopsy.*

Maude Orme, æt. 2, was attacked with vomiting and abdominal pain on Friday, April 14th, 1865; on the morning of which day her bowels had been freely moved. She continued to vomit incessantly until she was brought to me on Monday, April 17, but passed nothing by her bowels. The belly was flaccid, bore pressure freely, and no blood had been passed per rectum; no abdominal tumour could be felt. She was ordered a sedative mixture containing hydrocyanic acid, and also small doses of Dover's powder frequently repeated. Linseed-meal poultices to be kept constantly applied to the abdomen. On the 19th there was no improvement, and insufflation was practised, which seemed to give great relief. On the next day, the bowels were freely moved. April 28th. Since the last report the child has been decidedly better, and the bowels have been moved daily, but the abdomen is distended with flatus. She is feverish with a pulse of 100. May 3rd. Since yesterday there has been no motion; the vomiting has returned, and now is said to be stercoraceous. Abdomen distended. Insufflation repeated. May 5th. On the evening after the insufflation the bowels were moved twice, and since that three times, but the child continued very ill, and died on Sunday, May 7th. Post-mortem on Wednesday, May 9th. The abdomen was immensely distended with flatus, and decomposition advanced. On opening the abdomen, there was evidence of tolerably extensive peritonitis glueing together the intestines. On tracing down the small intestines, a point was reached, near the lower end of the ileum, where the distension ceased, and the remainder of the intestines were collapsed and empty. There were, however, no traces of invagination. The substance of the bowels, and the mucous membrane, were softened and showed marks of inflammatory action.

Remarks.—This case is interesting in many respects, and shows very well the effects of insufflation. The symptoms of invagination were well marked; the only prominent symptom that was absent being the escape of pure blood per rectum. It may be remarked, however, that this is not invariably present when the invagination occupies the small intestines, as it seems to have done in this instance; and, according to Rilliet and Barthes, it is not so valuable as a diagnostic symptom when the child has passed the earliest period of infancy. Insufflation was practised on two separate occasions, and each time the symptoms of obstruction disappeared, and the bowels were freely opened. Unfortunately, however, as not unfrequently happens, the original attacks gave rise to considerable intestinal derangement, eventually followed by an attack of peritonitis, which proved fatal. Indeed, before the second operation it is said that these vomited matters were distinctly stercoraceous—an event not common in children, but when it does occur almost necessarily of fatal augury. It may be doubted, therefore, whether it was altogether judicious to repeat the insufflation after symptoms of obstruction again showed themselves, although it seemed to offer the only prospect of saving the child's life, and its former success rendered us somewhat more willing to repeat the experiment.

CASE IV.—*Spinal Meningitis, followed by Paralysis.—Secondary Pneumonia.—Recovery.*

H. W., æt. 7 months, was brought to me on the 28th of November, 1864. He was taken ill on the 26th with heat of skin, feverishness, and vomiting. He now lies in his mother's arms with a pained and anxious expression of countenance, and constantly giving vent to a low moaning cry. His intelligence, however, does not seem to be in any way affected. The head is thrown back so as to bring the occiput almost into contact with the spine, and it is kept rigidly fixed in that position. The hands are tightly clasped, and his arms are quite rigid. Both legs and the left arm are motionless, and the mother observed the loss of power on the day after the commencement of the attack. The right arm is freely moveable, and sensation seems to be everywhere perfect. The child screams loudly whenever he is touched, so that it is impossible to ascertain whether there is any special tenderness on pressure over particular portions of his spinal column. The skin is warm. The pulse is 140, small and feeble. The bowels have been opened, and there

is now no sickness. He was ordered a mixture containing two grains of iodide of potassium to each dose, a teaspoonful three times a day, and a blister was applied over his spine. He was allowed a good nourishing diet of milk and beef-tea. A little brandy was subsequently prescribed. Under this treatment the infant materially improved, and on the 11th of December his head was retained in a natural position; the painful cry had ceased; the pulse had fallen to 100, and had become fuller and stronger; and he had commenced to use his right leg slightly. The next evening, however, he became worse, and is said to have had a convulsive seizure. I next saw him on the 13th when the respiration was rapid and panting, his skin burning hot, and the pulse much increased in rapidity. On auscultation, mucous râles were extensively heard over the base of the right lung, where there was also comparative dulness on percussion. Over the left lung there were numerous dry cooing râles with normal percussion note. We had now evidently to deal with a severe intercurrent attack of pneumonia, probably brought on by bringing the child backwards and forwards to the hospital in the inclement weather then prevailing. He was put on frequently repeated doses of sesquicarbonate of ammonia and chloric ether, with an increased amount of stimulant, and the mother was directed to envelope the chest in damped cotton wadding, covered with oil silk, which forms an excellent and easily managed substitute for poultices in young children. For many days the chest symptoms continued to be very severe, but eventually they diminished in intensity, and the child was noted to be breathing freely and easily, and the cough had almost disappeared. On the 3rd he was much improved in general health, took his food well, and was gaining flesh. There was still slight loss of power over his left arm. The left leg was completely paralysed, but the right could be moved easily. Sensation was apparently perfect, and the temperature normal. From this time the child has continued to improve steadily; and galvanism has been employed to the affected limbs with satisfactory results. He is now in good general health, but the power of motion in his left leg is still very imperfect, although better than formerly.

Remarks.—The symptoms in this case point to an attack of spinal meningitis, which, in all probability, was also accompanied by some inflammation of the substance of the cord; twice there was well-marked paralysis of the extremities. This does not, however, seem to have led either to softening or any other organic alteration of the nerve substance, since the paralysis was only temporary, and has been gradually disappearing as the

child recovered health and strength. Indeed, the prominence of spasm and rigidity of the muscles over other symptoms, render it probable that the principal seat of the attack was in the membranes of the cord, and the brain itself seems to have been almost entirely unaffected.

Most of the recorded cases of this disease have been in children considerably older than this infant, and Rilliet and Barthes state that they generally met with it in children from thirteen to fourteen years old. A considerable number of cases, however, are recorded occurring at a much earlier age, and one is mentioned in Dr. West's work in which the child was only five months old. The disease has frequently prevailed in an epidemic form, and in one epidemic of the kind which was observed in Ireland children under twelve years of age only were attacked. As regards the treatment, it may be noted that I adopted a less actively antiphlogistic plan than has been usually recommended, principally because the child did not seem to be in a state justifying depletion; nor did the result afford any reason for regretting that such a course had been pursued.

Dr. Murchison has quite recently stated his belief that the so-called cerebro-spinal meningitis is frequently only a modification or accompaniment of typhus,* and if this be the case, we have an additional reason for caution as to employing a very heroic plan of treatment.

Case V.—*Labour obstructed by pelvic tumour.—Cranioclasm.—Death.—Remarks on the comparative merits of Craniotomy and the Cæsarean Section.*

Mrs. McGyan, æt. 19. Primipara. I was called to see this patient one evening at 8 p.m. She had then been in labour about four and twenty hours, and on examination I found the pelvis blocked up by a firm but elastic swelling, smooth in its outline, and presenting no evidence of fluctuation. The enlargement was situated in the recto-vaginal cul-de-sac, and the finger could be passed behind it into the rectum. Nothing like a pedicle could be made out; as, however, the head had passed through the brim and pushed the tumour in front of it, no satisfactory conclusion could be arrived at as to this point. The obstruction was pretty uniform in all the pelvic diameters, but rather more in the antero-posterior, which was estimated to measure from $2\frac{1}{4}$ to $2\frac{1}{2}$ inches. The pains were strong, forcing,

* "Lancet," April 22nd, 1865.

and frequent. The pulse was 100, and symptoms of constitutional irritation were beginning to shew themselves. The fœtal heart sounds were distinctly audible below and somewhat to the left of the umbilicus. Although there was no apparent fluctuation a full sized trocar was passed into the mass, a precaution that should never be neglected in such cases, since tumours apparently perfectly solid have occasionally been found to contain fluid, and, after puncture, have collapsed sufficiently to admit of the passage of the child. This was not, however, found to be the case in this instance, and it was now evident that the only means of effecting delivery was either by the Cæsarean section or craniotomy. The latter alternative having been decided on, perforation was effected without difficulty, and the ordinary craniotomy forceps were applied, but failed to drag the head past the obstruction.

As much as possible of the vault of the cranium was now broken up and cautiously removed, but manipulation was extremely difficult on account of the narrow space which was left in the pelvic cavity, and after prolonged attempts to deliver with the ordinary craniotomy forceps and crotchet, lasting over more than two hours, it was found that absolutely no progress had been made. Finding my efforts unavailing, I desisted, and sent for Dr. Simpson's cranioclast, which I had not with me when I commenced the operation. One blade of this instrument was then passed within the skull, and the other external to the base of the occiput, the separate portions being then united like the ordinary forceps. By a twisting motion of the wrist I was enabled to fracture the portion of bone within the grasp of the blades, which were subsequently moved round to another part of the skull, where the same process was repeated. By a repetition of these manœuvres the entire base of the cranium was destroyed, the broken portions of bone remaining within the scalp, and subsequently moderate traction enabled me to effect delivery of the head, the shoulders and trunk afterwards passing without much difficulty. After delivery the tumour disappeared within the abdominal cavity, and could no longer be detected per vaginam. On examining the fœtal skull the summit of the cranium was found to have been entirely removed, and the occipital bone was fractured all round the foramen magnum, thus reducing the head to the smallest possible dimensions. Within twenty-four hours violent peritonitis set in, which resisted all treatment, and the patient sank on the third day. The friends would not allow a post-mortem examination.

Remarks.—It is unfortunate that the nature of the tumour

could not be ascertained after death, and there was no previous history of disease to throw any light on the subject. Had the patient been seen at an earlier stage of labour, it is possible that the tumour might have been pushed out of the way, as has often happened in such cases; but when I saw her it was quite immoveable, the head being well engaged in the brim.

Craniotomy was resorted to in accordance with the ordinary rule of British practice, there being sufficient space for the passage of a mutilated fœtus. On the Continent the Cæsarean section would, in all probability, have been preferred, and should I again meet with a similar case, I should have no hesitation in adopting that procedure. Even in osseous deformity, ranging between $1\frac{1}{2}$ and $2\frac{1}{2}$ inches, I believe that a careful examination of statistics will show that craniotomy is fully as perilous to the mother as the Cæsarean section. In these greater degrees of obstruction it is quite impossible to diminish the head sufficiently to admit of its passage without employing an amount of force which must necessarily be most dangerous to the mother. Added to this, the natural repugnance to destroy the fœtus which all must feel, too frequently leads to a delay which subjects the mother to the increased risk of a prolonged labour. Taking all cases of craniotomy together, Dr. Churchill estimates the mortality as 1 in 5, but if we had any means of ascertaining separately the results of those cases in which protracted efforts at delivery were necessary, it is highly probable that the proportion would be greatly increased. On the other hand, it has been shown that 81 per cent. of the cases in which the Cæsarean section was performed under favourable circumstances, that it is at an early period of labour, and before the patient was exhausted, recovered. This is in all probability a somewhat exaggerated estimate, but still it must be nearer the truth than that usually stated in this country, where the mortality forms no proper criterion as to the dangers of the operation, which has almost invariably been delayed until the patient has been reduced to a condition which would render her recovery from any operative procedure whatever almost hopeless. In a case of this sort, also, when craniotomy is performed, a tumour of low organization is necessarily exposed to an amount of pressure and contusion which must of itself be highly dangerous, and which would be entirely avoided were the Cæsarean section preferred. If the tumour were ovarian also we should then have an opportunity of removing it at the same time, and thus effect a radical cure without materially increasing the risk. In this instance, for example, although the precise nature of the growth was not

ascertained, it very probably was a fibro-cystic tumour of the ovary, and if so it might easily have been extirpated before the wound was closed.

We have here also a good example of the imperfection of the craniotomy instruments used in this country. Both the craniotomy forceps and the crotchet had been cautiously but perseveringly employed for fully two hours, but no progress was made, although, I believe, they had effected all they could have done, that is, broken up and removed the entire summit of the cranial vault. With Dr. Simpson's cranioclast I was enabled to destroy the resistent base of the skull in a very short space of time, and without any difficulty, and when this was effected the main obstacle to delivery was removed. This instrument seems scarcely to have received the attention which its simplicity and effectiveness merit, and it possesses many advantages over the ordinary craniotomy forceps. Even when the amount of obstruction is not great, and when it is not necessary to break up the base of the skull, it may be used as a simple tractor, after perforation, quite as effectually as any other instrument. The only other means of destroying the base of the skull is by the cephalotribe, which has not made any way in this country. Doubtless one of the lighter varieties, such as Braun's, would be found very effective in the ordinary cases in which destruction of the child is necessary, especially when the deformity is confined to the antero-posterior diameter of the brim, when there is usually ample space for the passage of the blades of the instrument by the sides of the ilia. But in a case of this sort, in which the obstruction is pretty equal in every direction, as much laterally as antero-posteriorly, it seems impossible to understand how the blades of even the smallest cephalotribe could be passed. No such objection could be urged against the cranioclast, which is not larger than the ordinary craniotomy forceps, and which possesses in an equal degree with the cephalotribe the great advantage of retaining the bones within the fœtal scalp, and thus avoiding the risk of lacerating the vagina, which is unavoidable when the bones have to be removed piecemeal.

MR. BAXTER ON MUSCULAR CONTRACTION.

(*Continued from page* 211.)

Enough evidence has been adduced, I believe, to show that the mere passage of an electric current along a nerve inversely is incapable of destroying the properties of the nerve throughout its whole course, and consequently this mode of paralysing the nerve cannot be adduced either in favour of or against the independence of the contractile property of the muscular tissue; the question therefore remains to be decided by other evidence. In discussing this question it is, as I have said in a previous part of this paper, important to bear in mind the two questions that are involved, viz., that of muscular contractility, and that of muscular contraction. When a nerve has been brought into the so-called anelectrotonic state, during the passage of the current, it may so happen that contraction of the muscular fibre may not occur upon stimulating it, or if contraction takes place, it is then supposed that the nerve has not been sufficiently paralysed. From my inquiries, it appears to me that during the passage of the current the muscular fibre may become permanently contracted during the passage of the current, and, consequently, no further contraction can take place; this permanent contraction, however, is not always obtained. Let the current be now suspended, tetanic contraction takes place. How is this? The nerve is now supposed to have recovered its normal condition, when we know that the trunk of the nerve has been completely disorganised, and that the effects may be due to a secondary electro-motor power established in the nerve, as shown by Matteucci, and which I have also been led to believe may depend upon the chemical changes which take place in the nerve under these circumstances.* When the muscular fibre, therefore, remains inaccessible to stimuli, this may arise either from a previous contracted state of the tissue or from a loss of its contractile power. It is a most important circumstance to bear in mind, that the experiments to elucidate this point have usually been made upon muscles separated from the body in which no restoration of the property can take place through nutrition. Again, if the contractile property be dependent upon the nerves, how can we account for the effect produced by elongating a muscle? Let a muscle be permanently contracted and inaccessible to stimuli, elongate it, and then apply the

* Edinburgh New Philosophical Journal. January, 1864.

stimuli, contraction will take place. I do not believe, in fact I know to the contrary, that the mere elongation of an exhausted muscle will not enable contraction to take place; some degree of contractile power must exist in the muscle. This brings me to speak of the elongation of a muscle. I believe the elongated state of a muscle to be the most important condition, for without it contraction cannot take place; it may be looked upon as the vital condition, and dependent upon two circumstances; first, upon that state which I have termed a polarized state, a state of tension due to nutrition; and, secondly, upon the antagonistic action of other muscles or elastic tissues. As the contractile power of a muscle bears a most important relation to its electrical condition, there is every reason for believing that this elongated state depends also upon the electric state, and consequently the electrical property of the muscular tissue becomes its most important property. This condition of the tissue is brought about and maintained by nutrition. According to this view muscular contraction must be considered as resulting from the absence of a counteracting cause, viz., that which produces the elongation of the muscles. Muscular contraction does not arise from the superinduction of any other property, but from the loss of a property (electricity), and consequently it is difficult to conceive how contraction can be brought about by the superinduction of any contractile power by means of nervous agency. In further confirmation of these views I may just remark, that during muscular contraction we can obtain evidence that electricity is evolved, and consequently there is a loss of power.*

It is a well-known fact that after a muscle has been fatigued, contractions are more easily excited. It may be asked, How is this? I believe these results may be in a great measure referred to the absence or weakness of the antagonistic action of the other muscles; for instance, let the gastroc nemius muscle of the leg be excited to contract, with occasional intermission through the nerve, after a short time the contractions may more readily occur upon stimulating the muscles; the extensor muscles of the foot being comparatively weaker than those of the flexors (the gastroc nemii), have not that power to prevent or resist the contraction of the latter. This fact opens out a most important question in regard to the physiology of the nervous system, viz., how far those effects which have been referred to the controlling power of the nerves, may not be due more to the antagonistic action of muscles than to any real action of the nervous

* *Heat*, acting as a repulsive force, must not be overlooked. Heat as well as electricity is evolved during muscular contraction.

system, and what has been generally referred to as a paralysis of a nerve, may in fact be due to the overpowering action of one set of muscles over those of another set.

I need scarcely refer to the fact that contractility is a property not limited to the muscular tissue; or even is the existence of the nervous system required for its manifestation, for we find plants to possess contractile powers in their tissue.

We are now brought to the consideration of the question, that if the contractility of the muscle be a property of the tissue, independent of the nervous system, in what manner does nervous influence cause contraction? Since it has been shown that electricity is associated with the nervous tissue, it has been naturally supposed that this electricity of the tissue must have an important influence in muscular action; that currents of electricity are developed during nervous action, which react upon the muscular tissue, and thus cause contraction. My own investigations have led me to the conclusion that it is not through the medium of the electricity of the nerve that nervous action takes place; nerve force is a force sui generis, capable of reacting upon electric force, but does not generate it. Nerve force reacts upon the electric force of the muscular tissue, in the same manner as it reacts upon the electric force in the electric organ of the fish; and during nerve action electricity is evolved in the latter as well as in the former, when it is also accompanied with muscular contraction as one of the resultant effects. Although a most intimate connection exists between nervous power and muscular power, we cannot say that nerve force is converted into muscular force (electricity), inasmuch as the muscular power pre-exists, and is independent of nervous power, hence contractions may arise independent of nervous agency.

It would be extremely desirable if we could obtain some means of estimating nervous work in the same manner as we can muscular work. I know of none at present; I have endeavoured to ascertain whether the electric state of the nervous tissue might not afford some evidence, as it is the only property connected with it which is capable of being measured, if we except the conducting power of the nerves in transmitting impressions, but have failed in obtaining any decisive evidence. Although muscular power bears a relation to nervous energy, still as the former is independent of the latter, this also fails; how far the variable constituents of the different secretions and

* Philosophical Magazine. July, 1862.

excretions may lead to the solution of the problem remains to be seen.* The measurement of the mental work appears to me to present the greatest difficulty.

ON THE MODERATING CENTRES OF THE REFLEX FUNCTION OF THE SPINAL CORD.

By Alexander Herzen, M.D.

The fact that the destruction of the brain, or its mere separation from the rest of the nervous system, causes a considerable increase of the reflex movements in the whole body has been known for many years. (It has been observed in the case of the human subject and in animals after decapitation.)

This fact has been explained in different ways: in 1858, Prof. M. Schiff gives, in his "Treatise on the Nervous System," the following explanation, based upon a great number of experiments: any considerable part of the nervous centres (brain or cord) being taken away, or its communication with the rest interrupted, the outer irritation that provokes the reflex movement is prevented from spreading to the whole nervous system, and it, therefore, concentrates itself upon that part of it which remains in its normal relations, and forces it to respond with a more violent reaction. Other physiologists do not admit this explanation, and think that the brain has a special influence, exerting a kind of moderating influence over the reflex function of the spinal cord, and that the increase of reflexion after the destruction of the brain, arises from the absence of the moderating centres contained in it.

But up to the year 1863, no one had tried to demonstrate this theory by the only positive method in matters of physiology —by experiment. At last Mr. Setchenoff (Prof. of Physiol. at St. Petersburgh), published a memoir "On the Moderating Mechanism of the Reflex Function of the Spinal Cord, in the Brain of the Frog" (Berlin, Hirschwald & Co.); and Mr. Matkiewicz published, in Henle and Pfeuffer's "Journal for Rational Medicine" (1864, 3d fascicle, xxi. vol.), a very laborious memoir "On the Action of Alcohol, Strychnine, and Opium upon the Moderating Centres of Reflex Action in the Brain of the Frog." A full critical analysis of these memoirs is contained in my work upon this subject (which may be had

* I would refer to the investigations of Haughton and others in regard to the development of urea during mental and bodily work.

at Messrs. Trübner & Co.'s, Paternoster Row). Here I shall only indicate Mr. Setchenoff's experimental method, and give his results.

He opens the skull of the frog, uncovers the brain, makes transverse incisions through it at different heights, and observes the effect produced by each incision upon the reflex movements of the posterior extremities of the animal. He considers every incision as a strong mechanical irritation of the points it passes through, and obtains accordingly a great decrease of reflexion whenever it falls upon certain points, which he, therefore, considers as the moderating centres in question. When the incision falls behind those points, he obtains a great increase of reflexion—for, as he thinks, it then interrupts the communication of the moderating centres with the spinal cord. He measures the reflex power of the cord by immersing the posterior extremities of the frog in a very dilute solution of sulphuric acid, and notes the time that intervenes between each immersion and the first reflex movement of the animal.

The following are his results:—

I. Sections through the hemispheres: no effect; that is, the reflex action only becomes a little more regular.

II. Section through the thalami optici: complete cessation of reflex action for five or ten minutes.

III. Section through the corpora quadrigemina: the same effect.

IV. Section behind the corpora quadrigemina: increase of reflex action, beginning one or two minutes after the incision.

V. Section at the point of the fourth ventricle: increase of reflex action which begins sooner.

VI. General result: The moderating centres are situated in the thalami optici, the corpora quadrigemina, and, perhaps, also in the medulla oblongata.

Mr. Setchenoff repeats his series of experiments with chemical instead of mechanical irritation (by means of common salt to his different sections), and comes to the same results.

The question is certainly one of great interest and importance. Having found all the requisite means for work at the Museum at Florence, I decided upon repeating Mr. Setchenoff's experiments under Prof. Schiff's guidance, and, as Prof. Schiff had already noticed previously that also peripheral nerves exert a certain influence upon the reflex action, I extended my researches to the brain, spinal cord, and nervous trunks. A full description of thirty-five examples of my experiments will be found in my memoir. My results are the following:—

I. Irritation of any considerable part of the nervous system, central or peripheral, produces a great decrease of reflex action.

II. Destruction of any considerable portion of the central or peripheral nervous system produces a great increase of reflex action in all the other parts of it.

III. General result: The moderating centres do not exist.

ON THE DEVELOPMENT OF MORBID BONE.

By Dr. ROUDNEFF, Assistant to the Pathological Anatomical Institution of the Medico-Chirurgical Academy of St. Petersburgh.

THE controversy concerning the development of normal bone, which has been carried on recently between Lieberkühn and H. Müller,* led me to investigate the process development of bone in different cases of disease. It is true that the pathologico-anatomical school had always taught that morbid bone was developed from connective tissue, as well as from cartilage.† But the laws of the formation of the tissues are the same in pathological as in physiological cases. If then the pathologists say that cartilage can be transformed into bone,‡ are they right in reality? On the other hand, are not the physiologists wrong who state that, in the physiological state, there is no case of direct transformation of cartilage into bone?§ In this investigation I limited myself to the examination of morbid bone, which I have met with either in form of growths both in man and animals, or in form of ossifications of the normal cartilages of the ribs, larynx, &c.

ENCHONDROSES.‖

These are the so-called cartilage tumours, which spring from the normal cartilage, the formation of which is consequently

* *H. Müller*, Ueber die Entwickl. des Knochens, 1858; *Lieberkühn*, Arch. of Anat., 1862, s. 702; *H. Müller*, Würzbürger Naturwiss. Zeitschr. 1863, Bd. IV. Hft. 1; *Lieberkühn*, Arch. of Anat., 1863, Hft. V.

† *Förster*, Atlas d. Microsc. Path. Anat., Plates II and III.

‡ *Virchow*, Cellularpath, 1862, s. 391.

§ In the skull bones of the frog every stage of the process of the formation of normal bone can be demonstrated. The cartilage is directly and immediately converted into bone. See my drawings and observations, in the lectures given at the Royal College of Physicians, 1861, German edition, pp. 130, 131.—[EDITOR.]

‖ *Virchow*, Die krankh. Geschwülste, 1863, s. 438.

connected with that of normal cartilage. Of many tumours of this kind, I have examined a very large one, which exists in the pathological collection of Würzburg. This tumour, which is about the size of the head of an adult, occupies the cartilaginous portion of the lower ribs. Its structure is hyaline cartilage, with very large corpuscles. The tumour contains very few blood-vessels, as is usually the case in cartilage. There were, however, dispersed through it, islands of cretaceous matter. After dissolving this matter in acid, there remained very little changed cartilaginous tissue. However, there were also true bone formations. In examining this tumour, there are two questions:—1. As to what changes occur in the germinal matter of the cartilage, when the bone is being formed. 2. The nature of the changes taking place at the same time in the hyaline tissue or formed material of the same cartilage. The first question is answered by all observers more or less unanimously. All authors adopt the conclusion that the osteal cells of the bone, which have replaced the cartilage, are the descendants of the cartilage cells. But concerning the changes occurring in the hyaline matter during the process of ossification, the opinions of authors differs much, and are even contradictory. Some say, that the hyaline matter of cartilage takes no share in the formation of the bone at all, that it undergoes regressive degeneration.* Others on the contrary, maintain that there exists a certain relation between the formed substance of the cartilage and of the bone.†

I will, in the first place, state the facts as they may be observed in morbid bone, and in the hope of throwing some light upon the consideration of the general question. In those cases which I have examined, the osseous tissue in the cartilage tumour partook of the characters of spongy bone. It consisted of more or less thick cross bars and plates of true bone, the structure of which was lamellated and exhibited many well marked lacunæ. Between the plates, young round cells, and some fat cells, surrounded by blood-vessels, were situated. On examining the cartilage, which was surrounding the osseous

* *H. Meyer*, Arch. of Anat., 1849, s. 292; *Bruch*, Beiträge zur Entwickl. d. Knoch., 1850, und Zeitschrift f. wissen. Zool., 1855, Bd. V. s. 145; *Schlossberger*, Die Chemie. d. Geweb., 1856; *Baur*, Arch. of Anat., 1857 s. 347; *Hoppe*, Virchow, Arch. Bd. V. s. 170; *H. Müller*, l. c.; *Kölliker*, Handb. d. Gewebel, 1863 s. 94, v. 258.

† *Rokitansky*, Lehrb. d. Path. Anat., 1855, Bd. l. s. 173; *Aeby*, Die Symphis. oss., pub. 1858; *Lieberkühn*, l. c.; *Lionel Beale*, Archives of Medicine, 1862, Vol. III., p. 83; *Virchow*, l. c.; *Robin*, Journal de l'Anatomie et de la Physiologie, par M. Robin, 1864, No. 5, p. 514, and Comptes Rendus et Mémoire de la Soc. de Biol., 1850, p. 119.

formations, I constantly found more cartilage corpuscles, than in other parts of cartilage further from the bone. The corpuscles were nearer to each other. The hyaline matter contained in many situations small masses of calcareous matter,—in short all the appearances were analogous to those which are observed in the epiphyses during the process of ossification. The chief difference seems to be the following: in the epiphyses the cartilage corpuscles, after proliferation, become arranged in regular rows.* In the morbid cartilage, before ossification, I did not observe such typical and regular arrangement of cartilage corpuscles. But the more or less regular position is, after all, not important. It is essential that the cartilage corpuscles multiply near the osseous border, and their division gives rise to many young corpuscles, which lose their original character and appear as indifferent elements. These are in a condition to take different forms by their further development.

Next let us consider what is going on at the same time in the hyaline formed matter. It does not remain without undergoing change; it does not perish by regressive metamorphosis. During the process of ossification, both germinal and formed matter are placed under entirely new conditions of existence, in consequence of which the character of the germinal as well as the formed matter should be altered. These conditions consist in the supply of a greater quantity of blood than before ossification, because the process of ossification is always accompanied with the development of blood-vessels in the cartilage. The hyaline cartilage alone retains its character without blood-vessels.† Of this I am convinced by investigation of normal as well as morbid cartilage. As soon as the blood-vessels begin to be developed in the cartilage, its nature becomes changed. The change in the germinal matter is, as I have above described, but the formed hyaline matter ceases to yield chondrin; by boiling it gives gelatine, that is, it has acquired the character of the formed matter of connective tissue. Afterwards I hope to show how I succeed in proving the truth of this statement by chemical means. But anatomical examination affords the chief ground for this opinion.

In disease, the direct transformation of hyaline cartilage into connective tissue often occurs,‡ for instance, if caries in a

* Bidder, Arch. of Anat., 1843 s. 372.

† I can quite confirm what Dr. Roudneff says upon this point. It would appear as if the distribution of an increased quantity of nutrient matter was connected with the changes in which the masses of germinal matter play so important a part.—[L. S. B.]

‡ *Führer*, Virchow's Arch., Bd., V. s. 129.

bone takes place near the cartilage, the latter often becomes fibrous, the more so if the tissue surrounding the cartilage becomes freely supplied with blood; and if the development of the blood-vessels does not cause, it seems to very much favour, the conversion of hyaline cartilage into fibrous connective tissue. The same alteration of cartilage takes place in the course of inflammation, for example, in arthritis. I have had occasion to observe all the forms and stages of ossification of cartilage in the hyaline enchondrosis of the ear of a rabbit. I injected the blood-vessels of this tumour in order to ascertain what the influence and meaning of blood-vessels in cartilaginous tumours may be. The tumour was the size of a hen's egg. It arose from cartilage of the ear, and a careful examination showed that upon both surfaces the tumour was covered with perichondrium. The cartilage of the tumour had partly the character of reticular cartilage of the ear, but mostly it was hyaline; the cartilage was partly converted into bone, partly into connective tissue, so that the whole tumour might have been regarded as an example of the mixed osteo-cartilaginous tumour. I made preparations for the microscope by filing and polishing small pieces. Another mode of preparation which I adopted was macerating pieces of the tumour in a solution of chromic acid (of 2 per cent.). By cutting slices in several directions, the blood-vessels being injected, I convinced myself that the character of different parts of tumour was due to the amount of blood which supplied them. I never met with blood-vessels in the hyaline matter, without its alteration at the same time. As soon as a certain capillary loop approached the cartilage, the latter became changed, so that the corpuscles began to be divided, and the transparent hyaline formed matter became fibrous. Thus it is clear that the cartilage can be transformed into fibrous tissue, and consequently we must admit the possibility of conversion of cartilage into bone, since the organic basis of bone is the same as that of fibrous tissue. In reality in the same tumour of a rabbit I have had occasion to study all the forms of development of pathological bone. It happens as follows, at the same time that blood-vessels arise the germinal matter of cartilage increases, and the masses begin to divide. The young elements thus resulting, having the form of cells, with numerous blood-vessels, are contained in round or oblong cavities, of which the cartilage consists at this period. The walls and spicules between these cavities consist of hyaline matter. The elements contained in the cavities do not remain a long time indifferent, some of them soon become fatty cells, and others are transformed into osseous corpuscles, while the

calcareous matter is deposited in the formed matter. These appearances of commencing ossification are to be seen always round the periphery of the above cavities, so that in a short time an osseous layer is formed round the entire cavity, and appears under the microscope like a ring. This ring then increases in thickness towards the centre, so that at length the whole of the cavity is filled with bone tissue. The entire closing of the cavity by bone is possible only if there are no blood-vessels, but mostly there is one, and in such a case the growth of the bone inwards ceases at an early period, and thus a cavity results, which is surrounded by bone, and contains the blood-vessels and fatty cells, with a little fibrous tissue: in one word, thus the cancellous tissue is formed. It is clear that the bone in this case is rising from the cartilage, even by transformation of the latter into the former, but not by thrusting it out, as H. Müller explains it. The cartilage is not abolished at all, the bone grows on account of it, and swallows it up, as it were, just as certain morbid structures, such as cancer, cause the removal and live at the expense of the subjacent fibrous tissue, from the germinal matter of which, perhaps, they may have originated.

The above-mentioned authors think that the bone is a new formation, and that it thrusts out the subjacent cartilage, and consider that the laying on of the osseous lamellæ occurs in a direction from within outwards, that is, that the new layers of bone, or new lamellæ are formed in the cancelli, consequently in every system of bony lamellæ the external lamellæ are the oldest, and those nearest to the centre of cancelli are the youngest; these latter remove the external ones more and more outwards, while the hyaline structure is absorbed, in consequence of the pressure of the osseous lamellæ. This opinion of the absorption of cartilaginous tissue during ossification was grounded upon the fact of the existence of a sharp border between cartilage and osseous tissue, but this sharp border is not constantly observed. On the other hand the impregnation of the nearest layer of cartilage with earthy salts in form of grains has been referred to, as if it proved the regressive metamorphosis of cartilage. Our investigations of pathological tissues have led us to form other views on the growth of the bone outwards from the loops of vessels. We agreee perfectly, that the formation of the bone depends directly upon blood-vessels, which are found in the Haversian canals or in the cancelli. The development and growth of each so-called partial system of osseous lamellæ is accomplished under the influence of these vessels. But if in reality the osseous lamellæ could

be rising only round the blood-vessels, consequently if the youngest of them should continue to remove those previously formed to the periphery, till the whole partial system were perfected, we should see under the microscope constantly the smooth border of the peripheral part of osseous lamellæ, but in reality this is never the case. The border of the system of lamellæ outward is always uneven, rugged, with more or less lengthy processes of irregular shape (Zaikenbildung, Pseudomorphosis of H. Müller), as if these processes grew in the adjacent cartilage layer. These appearances show clearly that the bone in the periphery of glomeruli grows independently, but it is not pushed from within outwards by the youngest lamellæ. I will add, that this independent growth is produced in consequence of changes in the adjacent cartilage. In the adjacent layer the proliferation of cartilaginous corpuscles is always observed, consequently this layer of cartilage, on coming into contact with the osseous tissue, is subjected to the same conditions, which obtain when the blood-vessels appear in the original cartilage. The corpuscles begin to be divided, they take the character of indifferent elements. The formed substance which yielded chondrin, now yields gelatin. But these alterations certainly take place at first around the cartilaginous corpuscles, therefore the first appearance of new bone takes place on the spot of the so-called capsules; thus the growth of osseous tissue progresses in form of processes or protrusions. Sometimes the deposition of earthy matter proceeds slowly, whilst the transformation of the cartilage into fibrous tissue is performed rapidly. In this case the new bone appears to be separated from the cartilage by a more or less thick layer of fibrous tissue. The cartilage is not absorbed, but it becomes bone. So also the ossification round the periphery of glomeruli takes place in the same manner as within, but with this difference, that here the proliferation of cartilaginous corpuscles is not so rapid as is the case in the cavities which first appeared. Concerning the sharp border between cartilage and bone, we would remark that this is by no means of any importance, and no argument can be based upon this fact against the continuity of the cartilage and bone. The sharp border is, in fact, caused by the different refraction of the rays of light in cartilage and bone. If such a border really existed, it would show that there was a limit to the growth of the bone, and the adjacent cartilage would perish from the pressure exerted by the growing bone. In this case, however, we should observe indications of the cartilage having been subjected to pressure. There would be flattening of the corpuscles, breaking up of the germinal matter

into granules, in short the appearances of regressive metamorphosis; but nothing like this is to be observed in the adjacent cartilage. On the contrary we find the corpuscles undergoing proliferation. Often we may demonstrate how one-half of the so-called capsule becomes bony, while the other remains cartilaginous. The sharp border does not always exist, and sometimes the cartilage refracts the light in nearly the same degree as bone, so that the line of demarcation between bone and cartilage cannot be observed at all. On examining such a specimen of cartilage by polarised light, I have observed the appearances of double refraction in the layer of cartilage, which was adjacent to the bone. It is certain that the cartilage before it becomes bone undergoes a certain change in its composition. Concerning the deposition of salts of lime in form of grains in the layer of cartilage near the bone, I am obliged to state that this deposition is not the sign of the death of tissue because the germinal matter is undergoing proliferation at the same time. The precipitation of calcareous salts, as an expression of the death of tissue, is never accompanied with growth or proliferation of germinal matter.* Consequently this deposition of calcareous matter may be more naturally explained as follows: the germinal matter in this layer of cartilage is already so altered, that it is able to make out the salts of lime, but the formed matter, in connection with which the salts must be coming, did not reach still the full transformation in the tissue yielding the gelatin, therefore the salts remain for some time in state of grains. The deposition of salts in the corpuscles themselves does not contradict our opinion; this seems to be analogous with many pathological appearances, for instance, the impregnation of the hepatic cells by pigment of bile, when the secretion of bile is retained.

But some will see the proof of the regressive metamorphosis of cartilage during the ossification in the arising of fat, which is to be observed in the cartilage. But this fat appears always in the cells in form of large drops: the cells usually are surrounded with blood-vessels, and arise from those of cartilage, consequently it is the progressive metamorphosis; this new formation of fatty cells is easily to be understood in this case after it has been proved, that the matter yielding the gelatin as well as albuminous matter give rise to the fat under the in-

* This remark, probably, refers to some observations of my own, but Dr. Roudneff seems not to be aware that I regard all tissue as having ceased to live. The living germinal matter may be undergoing proliferation, while the formed material, immediately surrounding it, may be lifeless and the seat of deposition of calcareous matter.—[Ed.]

fluence of oxidising substances, but yet in our case there are plenty of oxidising substances. In consequence of superabundance of blood, the transformation of cartilage goes farther than connective tissue, that is, the latter becomes fatty tissue. Whilst in the cases of regressive metamorphosis the fat appears always in form of small grains or free drops, never in form of cells. So also neither the deposits of lime, nor the sharp border between cartilage and bone, nor even the appearance of fat, do prove the imaginary abolition of cartilage during the process of ossification.

Enchondromata.

Strictly under this name are known the cartilaginous tumours connected with several tissues of body, without being connected elsewhere with normal cartilage (Heterology). I observed the cartilaginous tumours with bony substance in their composition, in the skin, in the mucous membrance of the windpipe, and in the muscles. The formation of bone occurred directly from the cartilaginous tissue, and sometimes entirely without any border between the bone and cartilage, and even without any proliferation of cartilaginous corpuscles, but entirely by means of transformation of the corpuscles of cartilage into those of bone, while the basis substance of cartilage became that of bone, after having been changed in its composition, and after having received the inorganic salts.

The Ossification of the Permanent Cartilages.

This takes place chiefly in advanced age, and affects the cartilages of ribs, those of larynx, &c. The position taken up by the ossifying process cannot be termed either p e r i p h e r a l or c e n t r a l. Blood-vessels make their appearance, and this causes the change in the cartilage. Whether they are developed from perichondrium, in superficial layers of cartilage, or in the deep layer, the cartilage always begins to be converted into the young connective tissue according to the direction of new vessels. Cancellous cavities arise, and these are filled with formative elements, from which, by and by, the osseous tissue forms itself so that the new bone at one side will come into contact with the tissue contained in the above cavity, but at the other side it will immediately border upon the normal cartilage; the latter becomes the osseous tissue in consequence of changes above described, namely, the multiplication of its masses of

germinal matter, certain chemical changes by which the formed matter becomes converted into tissue yielding gelatin, and by deposition of inorganic salts.*

If the cartilage in course of ossification is not destroyed and abolished, but really becomes changed into bone, as the anatomical examination proves to us, it is reasonable to expect that by chemical means we should be able to convert chondrin into gelatin. If such a reaction happened, the question concerning ossification would be considered as solved. But because the rational formula of the above bodies is unknown, and their chemical composition is hitherto so uncertain, that we cannot say whether they are simple chemical combinations or mixtures; at least, because the mode of getting these bodies by boiling is exceedingly difficult, as well as in respect of convenient purity, therefore the characterising of these bodies remains very uncertain. So we are compelled to distinguish the very similar combinations by the comparison of all known reactions, which these bodies give with several reagents. Since the time† of J. Müller, who at first distinguished chondrin from gelatin, there were many attempts to convert chondrin into gelatin, but unsuccessfully. The method of M. Schultze‡ by means of alkalies appeared unsatisfactory; much better was the method of Friedleben,§ by maceration of cartilage in mild solutions of hydrochloric acid. In my investigations I got the same results on operating with the other mineral acids as well as on digesting the cartilage in artificial gastric juice. But the following method of operating gave the most satisfactory results:—Knowing that the ossification of cartilage in the body is always preceded and accompanied by the growth of new blood-vessels, in consequence of which the oxidation in the tissues becomes intensified, I employed chloric acid ($ClHO_6$), one of the oxidising matters, in order to try if it was possible to convert the chondrin into gelatin. For this purpose, several cartilages from animals, as well as from the human subject, after having been cleaned, washed in distilled water, deprived of perichondrium, and cut into small pieces, were boiled with distilled water in a Papin's digestor for sufficient time, till there was formed a more or less thick decoction of chondrin. Then the fluid was filtered to separate the particles not dissolved. All reactions of chondrin were tried upon a

* Canton, Transactions of the Path. Soc. of Lond., vol. xii, p. 53.

† Annal. d. Phys. u. Chem., Bd. xxxviii, s. 295, 1836.

‡ Annal. d. Chemie u. Pharm., Bd. lxxi, s. 266.

§ Zeitschrift f. Wissenshaf. Zoolog., 1860, Bd. x, s. 20; and Wilckens, *ibid.*, s. 467.

part of this solution. Further, a certain quantity of solution of chloric potassium ($ClKO_6$) was added to the warm solution of chondrin, and afterwards a corresponding quantity of concentrated sulphuric acid. The fluid was warmed for a short time, the acid was neutralised with one of the bases, the salt of which could be washed out by means of spirit; the solution now was evaporated to dryness, the residue well washed with alcohol; thus there was obtained a white matter, which, with water, gave the opalised fluid or solution. The chemical characters of this solution did not now correspond with those of the preceding; we got no reaction of chondrin. The new characters acquired were as follows: in a cool place the solution became gelatinised like the glutin; the gelatin dissolved in water did not give the reactions with solution of alum, and with that of metallic salts, as nitrate of silver, sulphate of copper, nitrate of mercury, chloride of iron, &c. The organic acids—acetic, oxalic, and tartaric—as well as mineral acids, did not produce any precipitate. In short, there was no reaction of chondrin. On the other hand, the solution of tannic acid produced an abundant precipitate; chlorine-water gave also a considerable precipitate. A solution of ferrocyanide of potassium with some drops of acetic acid did not change the fluid. The above reactions and the negative results show that chondrin was absent and glutin was present in the fluid, which we examined. It remains for us now to prove by ultimate analysis that the chondrin, under the influence of oxidating matter really becomes glutin. Our opinion concerning the transformation of cartilage into bone, or, what comes to the same thing, the change of chondrogen into collagen during ossification by the process of oxidation, receives confirmation from the circumstance that the quantity of cartilaginous tissue in the animal body always corresponds to the activity of the oxidising process. Particular classes of animals, for instance, fishes, possess in their skeleton more cartilaginous tissue than is the case in that of mammalia, because the former breathe less than the latter. On the other hand, the cartilages of the birds become ossified very early in consequence of the rapid circulation and free absorption of oxygen. Moreover, cartilaginous tumours occcur in birds very rarely.*

[Although the author of this paper has not full command of our language, I have thought it better to make very few alterations. In a paper of this sort there is serious risk of destroying the author's meaning by modifying his language.—Editor.]

* Stannius, Arch. f. Anat., 1848, s. 408.

BLACK MASSES CONNECTED WITH THE LINING MEMBRANE AND VESSELS OF AN OVARIAN CYST.

By the late Dr. Charles G. Ritchie.

Miss B., æt. 40, had been complaining for seven months of symptoms referrible to the ovary. A multilocular cyst of the right ovary was diagnosed by Mr. Spencer Wells, and ovariotomy was performed on the 9th of May last. The operation was conducted in the usual manner. Towards its close, a little black mass, of about the size, shape, and consistence of a split pea was removed from the abdomen, but much attention was not paid to the circumstance at the time.

The patient sank on the third day with symptoms of septicæmia, and the body was inspected 32 hours after death. After having ascertained the condition of the pedicle on the right side, the hand was passed along the left broad ligament, to determine the state of the left ovary. No distinct ovary was to be felt, but in its place a pouch containing gritty particles.

On a more minute investigation, it was found that the left ovary had been converted into a cyst, capable of containing a small Normandy pippin. The wall of the cyst had an average thickness of $\frac{1}{8}$th of an inch, and, on being bent, crackled like parchment. The lining membrane was intensely black, and projected in folds into the cavity of the cyst. Posteriorly the wall had given way, and there existed a round smoothly-edged aperture $1\frac{1}{2}$ inches in diameter. The cavity of the cyst was empty as regards fluid, but to the lining membrane clung several little black masses similar to the one which had been observed during the operation.

The same black substances were found in Douglas's space. They varied in size from that of a bean to a mere pin point, the smaller ones being the more numerous, and from 20 to 30 may have been present altogether. They were hard, lustreless, and perfectly black, not unlike drops of black sealing wax. A portion of one of them was examined by Dr. Lionel S. Beale, and found to consist of inspissated and altered blood.

[The black matter in question exhibited no structure. After being softened in water, it appeared granular, and altered blood corpuscles were here and there to be detected, but I could find no crystals of hæmatoidin. The watery fluid filtered from the black deposit contained a little albumen.—L. S. B.]

A CASE OF PHTHISIS ACCOMPANIED BY DEVELOPMENT OF FUNGI IN THE KIDNEYS.

By Morris Tonge, M.A., M.D., Lecturer on Physiology at the Charing Cross Hospital.

(Plate XIX, Fig. 4.)

This case occurred at King's College Hospital, in the practice of Dr. Garrod, when his patients were temporarily under the care of Dr. John Harley. The singular appearances disclosed, on *post-mortem* examination of the kidneys, render it very interesting.

Jane F., æt. 45, a widow, following the occupation of a laundress, was admitted into King's College Hospital on February 12, 1864, in an extremely prostrate condition. With the exception of an attack of small-pox, she had escaped severe illness till about thirteen months ago, when she began to suffer from some symptoms of diabetes, the daily quantity of urine being large, her appetite ravenous, thirst excessive, and legs always more or less œdematous. There is, however, no evidence as to the state of the urine at that time. Since a slight attack of hæmoptysis about Christmas 1862, she had rapidly lost flesh, and a cough, which appeared about the same time, had become very troublesome. There was a slight hereditary taint of phthisis from the father's side.

On admission her face was much flushed, and she breathed hurriedly, the respirations being 40 or more in the minute. She was extremely weak, and the emaciation, though masked in the legs by slight œdematous swelling, was elsewhere very conspicuous. The opaque greenish expectoration she was constantly coughing up was rather viscid, and was got rid of with some difficulty. On percussion the whole front of the chest resounded less strongly than natural, and a patch of decided dulness existed below the left clavicle, where loud gurgling crepitation and breathing of a somewhat cavernous character were audible; gurgling was also very distinct under the right clavicle, whilst elsewhere was heard medium crepitation, varying slightly in character from point to point over the surface of the chest, and becoming daily coarser in quality. There was occasional nausea and flatulence, the appetite was indifferent, the tongue slightly furred, sleep disturbed, pulse 100. Trommer's test, applied to the urine on the third and fourth day after her admission, gave no evidence of the presence of sugar. No microscopical examination of the urine was made, and the mouth was not examined.

She gradually became exhausted, and died on February 19th, having been seven days in the hospital.

The *post-mortem* examination was made 26 hours after death, the weather being then extremely cold. The lungs, especially the right, were very adherent, and both were extensively diseased. Each, in its upper two-thirds, was studded with softening tubercles, many of them broken down into cavities of variable size, largest in the apex of the left lung. Towards the bases of the lungs the tubercles became more and more scattered, some of them enclosing microscopic cavities, while the extreme bases of the lungs were almost perfectly sound, though gorged with blood and serum.

The pelvis of the left kidney was very nearly filled with a yellowish white pultaceous substance, adherent to the apices of many of the pyramids. On detaching this pulpy mass, a ragged and somewhat softened surface of a yellowish white colour was left. The substance of the pyramid for a varying depth, averaging $\frac{1}{6}$th of an inch, was of the same yellowish white colour, and contrasted strongly with the dark crimson hue of the congested medullary tissue around, the line of demarcation between the two being quite abrupt. The diseased portions were of firm consistence except at the surface, and on the whole much resembled tubercular deposits. Two or three pyramids of the right kidney were affected similarly to those of the left, but to a very much slighter extent. The rest of each kidney appeared healthy. The gall-bladder contained twenty-five small facetted gall-stones.

Microscopical examination of the pulpy matter proved it to consist of the sporules and mycelium of a microscopic fungus, apparently a species of oïdium, possessing the following characters:—

1. Round or oval vesicles, single or grouped, containing one or more globules (probably oil globules or minute sporules), and sometimes granular matter.

2. Elongated vesicles, united at their ends so as to form continuous cylindrical tubes, branching dichotomously, sometimes terminated by strings or groups of round or oval cells, lateral development of such cells being also not uncommon. The tubes contained oil globules and granular material; here and there the contents had escaped, and an empty tube was seen. (See Plate XIX, Fig. 4, *a*, *b*.)

The white portions of the medullary cones were penetrated by the fungi for a varying depth, generally $\frac{1}{12}$th to $\frac{1}{6}$th of an inch.

If these fungi were dependent on fermentation, a supposi-

tion which the absence of sugar from the urine three days before death, and the absence of any history of a saccharine state of the urine render very unlikely, comparison of the somewhat large quantity of fungi formed with the very low temperature of the atmosphere during the short time intervening between death and the *post-mortem* examination, seems to shew that it must have commenced before death. The existence of the germs of the fungi in the blood, and their subsequent deposition and growth in the kidneys, is strongly opposed by the absence of similar deposits in other parts of the body where their presence might reasonably have been expected (*e.g.*, the lungs). The most likely supposition seems to be that they had crept up the bladder and ureters to the kidneys from aphthous patches, which may have existed about the orifice of the urethra, or from some parasite disease of the skin of the parts adjoining, their growth and extension being favoured by the low state of the patient's vitality shortly before death. As, however, there is no record of the presence of aphthæ about the vagina or elsewhere, or of any parasitic disease of the skin, the true source of the growth observed must remain mere matter of conjecture.

ON CHOLESTEATOMA.

By F. Bateman, M.D., M.R.C.P., Physician to the Norfolk and Norwich Hospital.

Tumours of the brain are usually attended by symptoms so obscure in their nature and diversified in their character, that in the present state of science we are unable to localize them with any amount of precision; their exact diagnosis is extremely difficult, if not impossible, and their treatment vague, uncertain, and almost empirical. It seems therefore extremely desirable to place on record all cases tending to elucidate this most interesting branch of pathology. The following case appears to me to be of some value, not only from the comparative rarity of the morbid growth discovered at the necropsy, but also from the satisfactory manner in which many of the symptoms observed during life could be connected with the lesions discovered after death.

On the 7th of July, 1866, Thomas Hook, a farm labourer, aged 26, a tall, well-made man, of considerable muscular development, was admitted into the Norfolk and Norwich Hospital under my care, with the following antecedent history. He had been married four years, had one child 21 months old, never

had syphilis, had always been a remarkably steady man, and had enjoyed very good health till about nine months after marriage, when the first morbid symptom shewed itself in a shaking, first of the right hand and then of the left, followed in a few months by frontal headache; these symptoms do not appear, however, to have interfered much with his pursuits, but about a year ago weakness of the legs set in, and inability to guide himself, his gait appearing like that of a drunken man, for whom he was often taken; and although his memory and intelligence remained unimpaired, it was about this time that some slight embarrassment in his speech was observed. He now for the first time sought the advice of an experienced surgeon, who writes to me to the effect that "he was struck with his bloated appearance, the glassy look of his eye, and his awkward, almost drunken gait, and that he complained of dizziness, general lassitude, and headache." He subsequently became an inmate of the Lynn Hospital, where he appears to have received some benefit; at all events, the frontal headache diminished considerably, but the relief in this symptom coincided with a dimness of vision, which resulted in complete blindness two months ago, the *right* eye being the first affected.*

Condition on Admission.—He can walk with assistance, but his gait is staggering like that of a drunken man, and he would fall unless supported; there is no actual paralysis of limbs, but perhaps a little less muscular power on the right than on the left side; sensation is unimpaired; there is difficult micturition, but no want of power over the rectum. He complains of severe frontal headache, is deaf on the *right* side, and is the subject of double amaurosis, being only just able to distinguish light from darkness; the pupils act, though sluggishly, and there is no apparent disease of the eye itself. The intelligence is unimpaired, his memory good; he converses coherently, with well-selected words, but he has an unnatural jerking spasmodic manner of speaking, as if the same want of power of co-ordination in the act of volition which is apparent in his gait extends to the expression of his ideas; his condition, however, is not that of incipient aphasia, for both the memory of words and articulation are good, but there is (if I may use the expression) ataxia of speech, as there is locomotor ataxia as applied to the condition of the limbs.

I have nothing to say about treatment, for having ascertained that various alteratives and counter-irritants had been

* The habit of smoking having lately been mentioned as a frequent cause of cerebral amaurosis, I may add that I have since ascertained that he was *not* a smoker.

already tried, I merely prescribed the hypophosphite of soda in gr. x doses, and a seton to the nape of the neck. After a few days the pain in the forehead increased in intensity, he became more drowsy, and on the eleventh day after admission, whilst conversing with a fellow-patient, he suddenly became livid in the face, and died instantly.

Necropsy, 13 hours after death. Body extremely well nourished, the fat of abdomen being an inch thick. Lungs slightly adherent at both apices, heart healthy, kidneys, liver, and spleen intensely congested. Brain congested, but extremely firm. The proper cavity of the arachnoid contained $5\frac{1}{2}$ oz. of cerebrospinal fluid; the ventricles were also dilated with fluid. On proceeding to remove the brain, a glistening tumour was found at the base of the cranium, adherent to and apparently springing from the posterior surface of the petrous portion of the right temporal, and from the basilar process of the occipital bones. The tumour was of the size of a hen's egg, although not exactly of that shape, for it was flattened from above downwards, its exact dimensions being $2\frac{1}{4}$ inches in its longest, and $1\frac{1}{4}$ in its shortest diameter; its surface was uneven, of a white, almost silvery appearance, resembling mother-of-pearl. It was invested with a membrane of the thickness of ordinary paper, which enclosed, as in a cyst, a substance of the consistence and appearance of wax, the central portion being of a dirty straw colour, not unlike tallow, but becoming paler, of an almost pure white, and firmer as it approached the circumference of the tumour. It had no adhesion except as above stated, and there was no trace of vessels in its structure. On observing closely the relations of this growth, it was seen to be lying against the under surface of the right lobe of the cerebellum, where an excavation existed for its reception; it exercised pressure on this lobe, also on the right side of the medulla oblongata, the right crus cerebelli, and the right side of the pons varolii in its whole extent. On submitting a portion to the microscope, numerous crystals of cholesterine and fat globules were observed, with a number of cells of a form and shape not familiar to myself or the other gentlemen who examined them with me. I therefore sent a portion to Dr. Beale, who pronounced the tumour to be cholesteatoma.

Before proceeding to comment upon the leading features of this case, I propose briefly to glance at the bibliography of cholesteatoma, and then to notice what are the points of resemblance and of difference to be observed in reference to this case, as compared with those of other observers. The first author who has given anything like a complete description of this growth

is Müller, who, under the name of laminated adipose tumour or cholesteatoma, describes it as a non-lobulated tumour, usually invested with a thin membrane, which forms a complete cyst, and composed of concentric layers of polyedrous cells, which have a lustre of mother-of-pearl. Paget classes it amongst the compound proliferous cutaneous cysts, the contents of which, he says, are always like the epidermal or other products of the skin, more or less perfect or degenerate. These tumours have more frequently been met with at the base of the brain, and most of the cases recorded have occurred in lunatics. In Beale's Archives, vol. i, p. 42, two cases, however, are mentioned where the growth was situated in the neighbourhood of the buttock; and Dr. Merriman, in an essay on tumours obstructing parturition, has given an account of one situated between the cervix uteri and the rectum, which, from the description he has given, was probably cholesteatoma.* Cruveilhier has given drawings of two cases which came under his notice; in both instances the tumour was developed in the brain.† He also gives the particulars of a third case as observed by Dr. Lepestre, of which I give the details, as tending to illustrate my own case.

A military man, aged 40, of a robust constitution, after 16 years' service, and several attacks of syphilis, was seized with a cephalalgia so violent and so obstinate that he was obliged to leave the army. He now became subject to giddiness, imperfect control over the lower limbs, soon resulting in complete paraplegia, with paralysis of the bladder and rectum. Frequent vomiting now occurred, his speech became embarrassed, signs of cerebral congestion shewed themselves, the upper extremities became paralysed, he was freely bled, and rapidly sank from exhaustion.

At the autopsy there was found considerable effusion into the ventricles and under the arachnoid covering the convexity of the brain, and on raising this organ, a tumour of about one-third the size of the cerebellum was found at the base of the brain, pressing on the left side of the pons varolii and of the medulla oblongata, and upon the anterior edge of the cerebellum, where was an indentation to receive it. The growth was described as of an even surface, of a pearly lustre externally, and on section consisted of concentric layers of a white substance, without trace of vessels, and resembling adipocere. But little is known of the genesis of these growths; when developed in the brain they are generally supposed to spring from the dura-

* Medico-Chirurgical Transactions, vol. x, p. 62.
† Anatomie pathologique, livr. ii, pl. 6.

mater; Rokitanski, however, hints that they may spring rather from the parietal layer of the arachnoid.

Having briefly reviewed the recorded cases of other observers, I would now direct attention to certain symptoms in the history of my own case, which seem to me of physiological and pathological interest.

1st. I think it may be inferred that the commencement of the morbid growth dates at least as far back as nine months after the patient's marriage, and the question naturally suggests itself how far the physiological change from the single to the married state contributed to the setting up of diseased action, for it is presumable that the cerebellum was the first organ whose functions were impaired by pressure. Now we know that in the phrenological system it is supposed that the cerebellum is that part of the encephalon which presides over sexual function, and from the experiments of Flourens, who has removed the cerebellum from pigeons in successive slices, it appears that animals deprived of this organ are in a condition very similar to that of a drunken man, as far as relates to their power of locomotion; without being paralysed there is an inability to accomplish any regular or definite movement, in fact they are unable to produce that combination of action in different sets of muscles which is necessary to enable them to assume or maintain any attitude. Like my patient Hook, they cannot stand still, and, in attempting to walk, their gait is tottering and unsteady. Dr. Todd mentions the case of a young army surgeon in which this defective power of coordination existed, and the patient staggered to such a degree that he was put under arrest on suspicion of drinking to excess; there was no distinct paralysis, and while in the recumbent posture he could move about his limbs freely. After a time he became amaurotic and comatose. The *post-mortem* examination revealed softening of the left crus cerebelli and corresponding restiform body, there was also a recent deposit of lymph at the base of the brain around the optic commissure.* Trousseau in quoting a case in which, without paralysis of sensation or of motion, there was the same indication of cerebellar disease, uses the term "cerebellar ataxia."†

2ndly. The occurrence of amaurosis and deafness is of great interest, as a minute examination of the tumour and of the surrounding parts enabled me very satisfactorily to account for these symptoms, for the anterior extremity of the growth pres-

* Cyclop. Anat. and Phys., vol. iii, p. 722s.
† Clinique Médicale, tom. ii, p. 533.

upon the posterior and inferior surface of the right thalamus opticus, and consequently upon the origin of the optic nerve, and the fact of the decussation of the fibres of this nerve at the commissure being only partial will explain the circumstance of the amaurosis being double. The deafness was quite as easily explained, for pressure was exercised on the portio mollis as it winds rounds the corpus restiforme, and also at its entrance into the meatus auditorius internus.

3rdly. It will be observed that the intellect was unaffected, thus differing from many of the cases recorded, in which the patient was lunatic; I have, however, been informed that there was strong hereditary predisposition to mental derangement—that his grandfather was subject to paroxysms of nervous excitement, and ultimately died by his own hand—that a paternal aunt was idiotic, and that a brother (deceased) was the subject of epilepsy.

4thly. It seems a matter for surprise that a tumour pressing not only on the cerebellum, but on the pons varolii and medulla oblongata, should have attained such dimensions before causing death. In reference to this point Wedl* speaks of these tumours attaining the size of a pigeon's egg, and Rokitanski† has seen them as large as a bean or a hazel nut, but I find no instance recorded where cerebral tumours of this description have been observed as large as that met with in Hook's case, although Müller mentions a specimen in the Berlin Museum *nearly* as large.

5thly. The embarrassment of speech calls for remark, more especially as lesions of that faculty are just now attracting so much notice. It is clear that the faculty of language was in no way affected—there was no amnesia, no disorder of ideation, but simply ataxia of articulation, probably produced by pressure on the origin of the glossopharyngeal and hypoglossal nerves. A French writer, Dr. Ladame, has published in the Gazette des Hôpitaux for June, 1865, an analysis of 332 observations of tumours of the encephalon, in which it appears that lesion of speech is by no means a common result. According to his table, lesion of speech is very rare in tumours of the cerebellum, but more frequent when the growth affects the pons varolii, the proportion being 6, 4 per cent. in the former, and 31 per cent. in the latter.

* Pathological Histology, p. 474.
† Pathological Anatomy, vol. iii, p. 335.

DESCRIPTION OF CERTAIN CASES OF TUMOURS OR ENLARGEMENTS OF NERVES (NEUROMATA), MOSTLY CONTAINING CAVITIES, AND ONE BEING OF UNUSUAL SIZE.

By John W. Ogle, M.D., Physician to St. George's Hospital.

Plate XIX, Figs. 1 & 2.

Having lately had the opportunity of minutely examining several specimens of tumours of nerves existing in the St. George's Hospital Museum, I am induced to place on record a somewhat detailed description of a few of these preparations.

Case I.—By far the most important specimen shows a neuromatous tumour, measuring about 6 inches in length by 4 inches in width, connected with and embedding the sciatic nerve and its posterior tibial branch.* It is oval in shape, and nodulated on its outer surface (see Plate XIX, fig. 1), and when recent, was enveloped by a dense fibrous capsule. It contains a large central cavity capable of holding an ordinary sized orange, with soft and shreddy walls (see Plate XIX, fig. 2). The great mass of the tumour is very firm in texture, and when cut into, after immersion in spirit for many years, was of a pale yellow colour, but towards its central part or cavity its substance is, in many places, much softened and very friable. In addition to the main trunks of the nerve which are embedded in it, (portions of which are plainly visible at the inner surface of the central cavity,) numbers of nerve filaments of various sizes may be seen separated, radiating, and spread out in a fan-like manner on its external surface (see Plate XIX, fig. 1). Moreover, for about one inch and a half above the tumour the nerve is much thickened and indurated.

Microscopical Examination.—The chief mass of the tumour was found to consist of the following elements. *a.* Fibrous and connective tissue of more or less firmness and solidity; *b.* Granular amorphous material; *c.* Round and oval cells of the size of pus globules for the most part, some being of larger size, swelling out and becoming clearer on the addition of acetic acid; *d.* Elongated fibre-cells and connective-tissue corpuscles; and *e.* Remnants of old nerve-cells, especially where nerves were seen by the naked eye.

When recently examined, the central cavity contained a quantity of semi-fluid broken down substance, which ran out when an opening was made into it.

* See the Hospital Pathological Catalogue, Series viii, No. 172.

Of this neuromatous tumour nothing is known, except that the leg was amputated during life. It was presented to our museum by the late Sir B. C. Brodie.

In addition to the interest which attaches to this specimen by reason of the large dimensions of the neuromatous tumour (Romberg speaks of neuromata attaining "the size of a hen's egg or more"), it is remarkable owing to the softening and breaking down which occurred in its structure, leading to the formation of the large hollow cavity. I neither know of any neuromatous growth of such a large size in any of our London Pathological Museums, nor yet of any such being placed on record, nor of any in which such complete central disintegration had occurred. In connection with this specimen, I may mention that Mr. Sibley brought a case of neuroma before the Pathological Society in 1857 (Feb. 17), in which "the central portion was found to be softened and broken down;" and in 1855 (Feb. 6) Dr. Van der Byl brought before the same Society specimens of neuroma of the posterior tibial nerve, in which one of the tumours contained "a small irregular cavity." Again, in 1859 (Oct. 18), Mr. Shillitoe presented to the same society a case of neuroma of the musculo-spiral nerve, enclosing a "cyst filled with serosanguineous fluid and clots of blood." Romberg also quotes a case of neuroma removed during life, and subsequently examined by Remak, in which a cavity existed in the interior, "of spongy texture, and traversed by fibrous trabeculæ, and filled with a yellowish-green serous fluid." The preparation was deposited in the Berlin Museum.

Case II.—This specimen was also presented to the St. George's Hospital Museum by the late Sir B. Brodie, and consists of a small rounded tumour in connexion with a nerve of the skin above the patella. It was removed during life owing to the great pain which it occasioned. On microscopical examination it was found to present the ordinary elements of white fibrous tissue.*

Preparations III. and IV.—These are portions of nerve having small cysts, originally containing fluid, connected with them. In the one case the cyst appears to have been formed by a collection of fluid beneath part of the neurilemma;† in the other one the cyst (connected with the median nerve, and the fluid of which, when recent, was quite clear) was situated deeper in the nerve, and had one or two filaments of nerve-tissue expanded over its surface. In the latter case the cyst

* See Series viii, No. 154, in Hospital Pathological Catalogue.

† See Series viii, No. 152.

was removed from a young man, owing to excessive pain suffered in the neighbourhood of the affected nerve, and with entire relief to the patient.*

As regards the above specimens, we have no proof that the affection of the nerve was the result of any mechanical or other local injury; but one cannot help comparing such tumours of nerve with those bulbous enlargements to which nerves, after division, during amputations of limbs, or in consequence of other injury, are often liable. Of enlargements so caused we have in the Pathological Museum of St. George's Hospital several well-marked examples. One of these, a remarkable one, has been described by Cæsar Hawkins, Esq., in the "Medical Gazette," vol. i., p. 271, and shows an oblong tumour in connection with the divided extremity of a median nerve, which, with the radial artery and certain muscles of the fore-arm, had been divided by an injury.† In this case some enlarged branches of the musculo-spiral nerve had formed at connection with the lower part of the median nerve, and amply supplied its deficiency. We also have other specimens showing the union of divided nerves.

Case V.—This is one of enlargement and induration of the pneumogastric nerve at a point just above the off-giving of the recurrent laryngeal branch. The enlarged part presents a fusiform shape in the longitudinal axis of the nerve. On microscopical examination, I found this increase in the bulk to be owing to the addition of a fibroïd material and fibrous tissue to, and their incorporation with, the substance of the nerve.‡ The specimen was removed from a patient who had extensive carcinomatous disease of the œsophagus and lungs and of the neighbouring parts to which the pneumogastric nerve was closely adherent. What influence the diseased condition of the pneumogastric nerve had in causing death must be altogether doubtful, seeing that the lungs and other important organs were exceedingly diseased in addition. This question naturally occurs to the mind having in remembrance the well ascertained results which follow ligation, or division of the pneumogastric nerves during life, especially the pulmonary œdema and sero-sanguineous infiltration of the lungs.

* See our Hospital Path. Catalogue, Series viii, No. 153.
† See our Hospital Path. Catalogue, Series viii, No. 164.
‡ See our Hospital Path. Catalogue, Series viii, No. 181.

MICROSCOPICAL EXAMINATION OF A SMALL MOVEABLE SUBCUTANEOUS TUMOUR.

By W. B. Kesteven, F.R.C.S.

Plate XIX, Fig. 3.

This tumour was removed from beneath the integuments of the abdomen of a man. It was of about the size of a filbert, and consisted of a tough fibrous cyst containing a white curdy matter. This matter on being viewed under a one-inch object glass presented a granular and fibriloid appearance, which under higher powers resolved itself into distinct fibres, connective tissue, and adipose cells, most of which had the aspect of being effete and disintegrated. Together with these were fragments of capillaries and of the ducts of several glands, epithelial cells, granular cells, a few oil globules, and crystals of cholesterine.

This substance was submitted to the action of ether, potash, and acetic acid, with little difference to the characters presented under the microscope. After the treatment with ether it was slightly less distinctly fibrous. Acetic acid had the effect of making it less translucent, while the cells, and crystals of cholesterine became more distinct. Potash rendered the fibres rather more apparent. The action of carmine rendered more clear the several elements of its composition by deeply tinging some of the epithelial cells (these are more darkly shaded in the accompanying sketch, Plate XIX, fig. 3), rendering visible their nuclei and granular contents, at the same time the faint outlines of the effete adipose cells were contrasted the more strongly with the living cells that had absorbed the colour of the carmine.

NETWORKS OR PLEXUSES OF DARK-BORDERED NERVE-FIBRES.

By the Editor.

Plate XX, Figs. 1 & 2.

In this Plate I have given two new drawings, illustrating the manner in which networks of dark-bordered nerve-fibres are arranged in voluntary muscle, and upon the deep surface of a highly sensitive mucous membrane.

In Fig 1 the long elementary muscular fibres are seen partly separated from one another, so that under high powers the very fine nerve-fibres in the intervals between them can be traced and followed to and from bundles of dark-bordered nerve-fibres. (See also Plate XII.)

The division and subdivision of complex nerve-trunks, and the formation of nerve-trunks by the union of the resulting bundles, is well seen in Fig. 2.

I have already advanced these broad facts as favourable to the view that nerve-fibres do not terminate in free ends, but form uninterrupted circuits.—(See my Croonian Lecture, Royal Society, May, 1865, and "Medical Times and Gazette," February 2nd, 1867.)

ON MUSCULAR POWER.

By H. F. Baxter, Cambridge.

(Continued from page 212.)

On the Mechanical Power of Muscles exerted during Muscular Contraction.

The following inquiry on the mechanical power of muscles exerted during muscular contraction must be considered as forming a continuation of a previous investigation upon the same subject, the results of which have already been published;* but as they were obtained upon frogs during the summer months, I have taken the opportunity of ascertaining whether any and what differences might be observed during the spring months, during the spawning season. These results will form the subject of the present investigation.

The mode of proceeding and the precautions to be taken in these experiments, for the particulars upon these points I shall refer to my former paper. There was one circumstance, however, elucidated on that occasion which appears to me of some if not of great importance. I allude to an increase in weight and vascularity of those muscles which had been excited to contraction when compared with those that had remained quiescent. As an increase in the vascularity might be referred to the division of the nerve, I have in the present inquiry divided the sciatic nerve in both limbs; and not only that, but also applied a ligature around the tendon of each limb, under the supposition that the blood-vessels might have been occasionally included within the ligature, and so occasioned a congested state of the muscle, although every precaution was taken to avoid doing so. I may, however, state that when the blood-vessels were purposely included within the ligature, I have not found that any increased vascularity could be attributed to this cause; nevertheless, it is a circumstance not to be overlooked.

* Edinburgh New Philosophical Journal, October, 1863.

I feel it necessary to make an observation respecting the *weight* of the muscle. I have stated it to be heavier and darker in tint, or slightly heavier; these terms are too vague to define a fact which may be so accurately described as weight. The balance at my disposal did not enable me to ascertain the differences so accurately as I could have wished, and unless the difference amounted to $\frac{1}{2}$ or to a $\frac{1}{4}$ of a grain, I have employed the terms heavier and slightly heavier; the same terms have also been employed to indicate the tint of vascularity as darker or slightly darker.

In drawing up the present tables two more columns have been added, to indicate the weight of the liver and that of the ovaries respectively. These facts may not have any immediate bearing upon the question of muscular power beyond that of their relation to the general weight of the body. My object, however, in recording the weight of the liver is for ulterior purposes. I was so much struck with the smallness of the liver when compared with those observed during the summer months, that I regret now not having recorded the weights of the former series; and even on the present occasion I regret not having taken the weights of the testicles. Such facts as these properly recorded may perhaps become of some value hereafter, when we have to consider the relation which the digestive organs and functions, as well as those of the sexual organs and functions, bear to the vital and muscular powers of the animal.

In Table I will be found the results of 30 experiments, arranged according to the date when they were performed. The frogs, male and female, enclosed in brackets, were copulating up to the time of the experiment. In analysing these results, and comparing them with those obtained on a former occasion, a remarkable difference will be found. On the present occasion we shall find that the sum total shows that 3 drs. 23 grs. weight of muscle were capable of raising a weight equivalent to 188 oz. 4 drs.; in other words, 1 gr. of muscle could raise, in round numbers, 445 grs., whereas on the former occasion one grain of muscle could raise 608 grs. The total weight of the frogs *before* experiment was 34 oz. 3 drs. 53 grs., *after* experiment, 33 oz. 6 drs. 4 grs., indicating a loss of 5 drs. 49 grs.

In Table II will be seen the influence of *sex* upon the results, and not only that, but I have also so arranged them as to ascertain whether the exercise of the sexual function may not have some influence. The males have been arranged under two heads, *first*, those that were embracing the female at the time of the experiment, and which were obliged to be separated

Table I.

No. of Experiment.	Date when performed.	Weight of Frog *before* Experiment.	Weight raised.	Weight of Frog *after* Experiment.	Weight of muscle.	Sex.	Weight of Liver.	Weight of Ovaries.	State of Muscles *after* Experiment.
	March	oz. drs. grs.	oz. drs.	oz. drs. grs.	drs. grs.		grs.	drs. grs.	
1	24 1864	0 10 40	6 2	0 10 20	0 6	F.	7½	3 48	The muscle used was 1 gr. lighter than the other, and appeared to the eye smaller; no difference in colour.
2	...	0 6 30	2 6	0 6 20	0 4½	M.	5	...	Muscle used slightly heavier, and slightly darker in tint than the other.
3	...	0 5 40	2 6	0 5 35	0 3	F.	4	1 38	No difference in colour or weight.
4	...	0 8 8	5 2	0 8 2	0 7	M.	8½	...	No difference in colour, slightly heavier.
5	25	0 11 5	12 2	0 10 50	0 11	M.	15	...	Slightly heavier, no difference in colour; muscles dark-coloured.
6	...	0 6 48	5 2	0 6 40	0 6	M.	6	...	Slightly heavier, and of darker tint.
7	...	0 6 50	5 2	0 6 44	0 6	M.	7	...	Weight same, slight difference in tint.
8	26	0 12 45	7 2	0 12 40	0 8	F.	11	3 20	No difference in colour or weight.
9	...	0 8 0	5 2	0 7 55	0 6	M.	6	...	Slightly heavier, with spots apparently of ecchymosis.
10	...	0 10 20	4 6	0 10 14	0 6	F.	8	3 2	Slight difference in weight.
11	...	0 7 12	4 6	0 7 0	0 6	M.	11	...	A decided difference in tint, and heavier by ½ gr.
12	...	0 11 20	5 2	0 11 14	0 6	F.	9	4 0	Slight difference in weight and tint.
13	...	0 10 40	6 2	0 10 32	0 9	M.	8½	...	Slight difference in weight and tint.
14	28	0 13 0	7 6	0 12 35	0 10	M.	13	...	Slightly heavier, but of darker tint.
15	...	0 12 17	9 2	0 12 7	0 11	M.	15	...	Muscle used ½ a grain heavier and of darker tint.
16	29	0 9 3	8 2	0 8 5	0 8	M.	12	...	No difference in colour or weight.
17	...	0 10 15	7 2	0 10 1	0 8½	M.	12½	...	Slightly darker and heavier.
18	...	0 7 50	5 6	0 7 2	0 5½	M.	8	...	Slightly darker and heavier.
19	...	0 5 34	4 2	0 5 22	0 4	M.	4½	...	Slightly darker, no difference in weight.
20	April 1	0 5 3	4 2	0 4 58	0 2	F.	3½	1 66	Slight difference in tint, none in weight.
21	...	0 12 57	9 2	0 12 50	0 11	M.	13	...	Half a grain heavier, and of darker tint.
22	...	0 13 54	7 2	0 13 46	0 6½	F.	14	4 53	A quarter of a grain heavier, and of darker tint.
23	...	0 7 18	6 2	0 7 10	0 6½	M.	8	...	Slight difference in tint, none in weight.
24	...	0 6 46	4 6	0 6 42	0 3½	F.	5	2 6	Darker tint, but of same weight.
25	...	0 5 54	6 6	0 5 50	0 5	M.	8	...	Slightly heavier, but no difference in tint; muscles dark-coloured.
26	...	0 8 13	6 6	0 8 0	0 7	M.	8	...	Slightly heavier, but of darker tint.
27	2	0 9 9	3 6	0 9 5	0 5	F.	8	3 27	No difference in weight, slightly darker.
28	...	0 8 5	6 2	0 8 0	0 8	M.	10	...	Slight difference in colour, none in weight.
29	...	0 14 3	8 2	0 13 58	0 8	F.	12	4 44	Slightly heavier, but of darker tint.
30	...	0 10 34	9 2	0 10 27	0 9	M.	11½	...	No difference in weight, slightly darker.
		34 3 53	188 4	33 6 4	3 23				

(indicated in Table I by brackets); and, *secondly*, those that were caught separated and distinct, and continued so up to the time of the experiment. Of course it was impossible for me to ascertain whether the males had copulated with females before they were caught or not. Respecting the females, I did not meet with one that was not embraced by a male frog. In all the males the dark-coloured swelling upon the fore-limbs was observed.

Table II.

Males (copulating). *Females* (copulating).

No. of Experiment.	Weight of Frog *before* Experiment.			Weight raised.		Weight of Frog *after* Experiment.			Weight of muscle.		No. of Experiment.	Weight of Frog *before* Experiment.			Weight raised.		Weight of Frog *after* Experiment.			Weight of muscle.
	oz.	drs.	grs.	oz.	drs.	oz.	drs.	grs.	drs.	grs.		oz.	drs.	grs.	oz.	drs.	oz.	drs.	grs.	grs.
2	0	6	30	2	6	0	6	20	0	4½	1	0	10	40	6	2	0	10	20	6
4	0	8	8	5	2	0	8	2	0	7	3	0	5	40	2	6	0	5	35	3
9	0	8	0	5	2	0	7	55	0	6	8	0	12	45	7	2	0	12	40	8
11	0	7	12	4	6	0	7	0	0	6	10	0	10	20	4	6	0	10	14	6
13	0	10	40	6	2	0	10	32	0	9	12	0	11	20	5	2	0	11	14	6
21	0	12	57	9	2	0	12	50	0	11	20	0	5	3	4	2	0	4	58	2
23	0	7	18	6	2	0	7	10	0	6½	22	0	13	54	7	2	0	13	46	6½
25	0	5	54	6	6	0	5	50	0	5	24	0	6	46	4	6	0	6	42	3½
28	0	8	5	6	2	0	8	0	0	8	27	0	9	9	3	6	0	9	5	5
30	0	10	34	9	2	0	10	27	0	9	29	0	14	3	8	2	0	13	58	8
	10	5	18	62	0	10	4	6	1	12		12	3	40	54	4	12	2	32	54

Males (distinct).

No. of Experiment.	Weight of Frog *before* Experiment.			Weight raised.		Weight of Frog *after* Experiment.			Weight of muscle.	
	oz.	drs.	grs.	oz.	drs.	oz.	drs.	grs.	drs.	grs.
5	0	11	5	12	2	0	10	50	0	11
6	0	6	48	5	2	0	6	40	0	6
7	0	6	50	5	2	0	6	44	0	6
14	0	13	0	7	6	0	12	35	0	10
15	0	12	17	9	2	0	12	7	0	11
16	0	9	3	8	2	0	8	5	0	8
17	0	10	15	7	2	0	10	1	0	8½
18	0	7	50	5	6	0	7	2	0	5½
19	0	5	34	4	2	0	5	22	0	4
26	0	8	13	6	6	0	8	0	0	7
	11	2	55	72	0	10	7	26	1	17

In analysing the results recorded in Table II, some most important facts are elicited. In the first place, we find that the muscular power of the female is greater than that of the male frog, inasmuch as the total weight raised by the ten females was 54 oz. 4 drs., whilst that raised by the ten males (copulating) was 62 oz., but the weight of the muscles in the females amounted only to 54 grs., whilst that of the males (copulating) was 72 grs., indicating, in round numbers, that 1 gr. weight of muscle in the female raised 484 grs.; whilst 1 gr. of muscle in the male (copulating) raised only 413 grs., whereas on a former occasion, during the summer months, the equivalents were—in the male 1 gr. weight of muscle could raise 646 grs., in the female 1 gr. raised 579 grs.

But another important fact is elicited in regard to the male frogs. When we compare the muscular power of those ten male frogs that were copulating at the time of the experiment, with those ten that were not doing so, an important difference comes out. The total weight raised by the ten males (copulating) was 62 oz.; the total weight raised by the ten males (distinct, not copulating) was 72 oz. The weight of the muscles of the former amounted to 72 grs. whilst that of the latter was 77; thus showing that whilst in the ten frogs (copulating) 1 gr. could raise 413, in the ten non-copulating frogs, 1 gr. could raise 448 grs. Even in the latter frogs the power did not equal that of the female frogs, inasmuch as 1 gr. of muscle in the female could raise 484 grs.

As the exercise of the sexual function has such an important influence, especially in the male frog, over the muscular power of the individual, I was anxious to ascertain whether this influence continued *after* sexual intercourse, and also whether the muscular power of the female continued to maintain its supremacy subsequent to the embrace of the male frog.

In Table III will be found the results obtained upon nine frogs, three females and six males; originally there were four females. They were all caught on the same day (the 26th of March), and the three females, when caught, were each embraced with a male, but the fourth female, which was found dead on the following day, was embraced by two males, thus leaving one male separate and distinct. When brought home they were put into a vessel, a pail having holes in the bottom of it so as to create a free circulation of air, but on the following morning the males were found separated from the females and remained so up to the day of the experiments, which was nine days afterwards. And I will now take the opportunity of mentioning the importance of attending to the

mode in which frogs are kept when caught for experiment; the vessel in which they are kept should not be air-tight. I have frequently observed this fact, if the frogs are kept in a pail they become, in two or three days, weak and flabby; whereas if they are kept in a vessel in which there is a free circulation of air, they continue lively for a longer period, provided the atmosphere be not too dry. I believe in the former case, in consequence of the absence of a free circulation of air the frogs become partly asphyxiated, on account perhaps of an accumulation of carbonic acid gas in the bottom of the vessel. Whether this explanation be a correct one, may be open to remark, but of the importance of attending to the mode in which they are kept, of that no doubt can remain.

TABLE III.

No. of Experiment.	Date when Performed.	Weight of Frog *before* Experiment.	Weight raised.	Weight of Frog *after* Experiment.	Weight of Muscle.	Sex.	Weight of Liver.	Weight of Ovaries.	Weight of each Testicle.	State of Muscles *after* Experiment.
		oz. drs. grs.	oz. drs.	oz. drs. grs.	drs. grs.		grs.	drs. grs.	grs.	
1	April 4	0 9 3	4 6	0 8 55	0 9	M.	11	...	2½	A ¼ of a grain heavier, and of darker tint.
2	...	0 14 13	4 2	0 13 49	0 13	M.	21	...	4½	Heavier and darker.
3	...	0 11 6	3 2	0 10 30	0 7½	M.	10	...	2	A ½ of a grain heavier, and darker.
4	...	0 13 37	5 2	0 13 30	0 8	F.	10½	4 48	...	Heavier, and of darker tint.
5	...	0 11 30	6 2	0 11 20	0 6½	F.	14	3 54	...	Slightly darker; of same weight.
6	...	0 8 30	4 2	0 8 20	0 4	F.	7	3 13	...	Slightly darker, and heavier.
7	...	0 4 3	1 6	0 3 58	0 3½	M.	5	...	½	Slightly darker, and heavier.
8	...	0 5 3	2 2	0 4 58	0 4	M.	5	...	½	Slightly darker, and heavier.
9	April 5	0 7 32	6 2	0 7 23	0 7	M.	8½	...	1½	Slightly heavier, and of darker tint.
		10 4 37	38 2	10 2 43	1 2½					

From the results recorded in Table III, we find that in the three females 18½ grs. of muscle could raise 15 oz. 6 drs., which corresponds in round numbers to 1 gr. of muscle raising 408 grs. Whereas in the six males 44 grs. of muscle raised 22 oz. 4 drs., which is equivalent, in round numbers, to 1 gr. of muscle elevating only 246 grs. But one of these males (No. 9) exceeded the others in power, its equivalent being 1 gr. of muscle to 444 grs., which exceeded the weakest of the females (No. 4).

To what circumstance can this great loss of muscular power on the part of the males be due? Had it been entirely due to the confinement and change of situation, the muscular power

of the females ought to have been affected in the same way; but this was not the case. They appear to have still retained the supremacy of their muscular power, although perhaps not equal to the other female frogs recorded in Table I. And again, the male frog (No. 9) which was kept a day longer was the most powerful of the males. There can be no doubt that all the frogs (both male and female) suffered in their muscular powers in consequence of the change of situation and confinement; but I am inclined to refer the great loss on the part of the males to be consequent on the exercise of the sexual functions producing a general loss of power on the part of the male. The males, when separated from the females, although kept with them, were apparently not disposed, or, perhaps, were incapable of renewing the act of copulation. Whether the male (No. 9) had so far recovered its powers in consequence of the delay (24 hours) that occurred in the experiment, or whether he had exercised the sexual functions and recovered before caught, would be difficult to decide. The importance of the general results indicating such a loss of power on the part of the males, I think cannot be denied. *A priori*, one might have been led to suppose that these effects might naturally occur, but perhaps not to the extent they are found to do; the fact, however, comes out much stronger and more prominent as the results of experiment than one could fairly have anticipated.

I will just make a few observations upon the general conditions of the frogs when caught, and previous to experiment. The males appeared dropsical, but the fluid was confined to the subcutaneous cellular tissue, like anasarca, but there was not a flabby condition of the muscles, and after the frogs had been kept some time, as those recorded in Table III, they appear to have thrown off the fluid by perspiration. The females presented in this respect a strong contrast to the males, they did not appear at all anasarcous; whether this was due to the swelling and distension of the ovaries absorbing the fluid, or by the distension of the body preventing the absorption of fluid taking place, may be questionable. I was much struck, however, at the absence of the extreme irritable condition of the muscular fibre, producing a tetanic condition, which I have observed on former occasions; whether it was owing to the experiments being performed earlier, and the great irritability occurring after the period of spawning, just before the frogs reappear, or whether it was due to the coldness of the season, snow, hail, and easterly winds prevailing, I am not prepared to say. That great variations in the conditions of the muscular system of the frog depend upon the season is now shown by these experi-

ments; hence we can obtain some clue to the apparently contradictory results that have been published in regard to the muscular irritability of the frog, and the caution requisite in forming hasty conclusions. Let any one, for instance, repeat the experiments recorded in my former paper, and contrast them with the results now obtained; he could come to no other conclusion than that experiment entirely failed to prove the results then obtained, in short would contradict them, as shown in this paper.

Respecting the state of the digestive organs, the stomach was empty and contracted; small nodules were occasionally seen in the small intestines, and a mass, generally, in the caput coli. The liver was dark-coloured, and the gall-bladder distended with dark-green bile; on two occasions only the liver was of a pale-yellow colour.

With regard to the condition of the muscle after experiment, the most important fact comes out that the muscle experimented on, and which has been used, tends to become heavier and more vascular than the muscle which has not been used; all my previous experiments, and those recorded in the present paper, with the exception of the first that is recorded, confirms this conclusion. With regard to this exceptional case, thinking at the time that the smallness of the muscle and diminished weight might be due to the state of the frog at the time of experiment, and also as a result of the experiment, I did not sufficiently examine the limb to ascertain whether an injury might not have occurred to the frog at some time previous, having occasionally met with united fractured limbs, in which one limb had become shorter and smaller than the fellow limb; it did not strike me until I had gone through three or four experiments that this might have been the case, and having thrown the frog away I could not re-examine it. With this exceptional case all the experiments tend to the conclusion that an increase in the weight of a muscle takes place during muscular action. It cannot be due entirely to the division of the nerve, as the experiments recorded in the present paper will show, for it does not take place with its fellow limb when subjected to the same conditions, excepting the excitement of muscular action. The increase in weight and vascularity depends upon the time the experiment continues, the weight that is made use of, and the power of the muscle. The weight must not be too heavy to overstretch the muscle nor to exhaust the power of the muscle rapidly, but the more prolonged the muscular contractions, provided they be not too frequently excited, the greater is the effect. This unexpected

conclusion is nothing more nor less than is exemplified every day. Let a set of muscles in the human being be in continued action, such as may be observed in the blacksmith's arm, or those in training for aquatic sports, such as rowing, or in pedestrians who take long walks, we soon see the effect that is produced in the size and bulk of muscles that have been thus brought into action, and we soon see also the converse effect when these exercises have been given up or intermitted for a time in the gradual lessening and diminished size of the same muscles. There can be no doubt that an increased action of a muscle is accompanied with an increased nutrition. I do not, however, believe that the making and unmaking, the formative and the disintegrating processes takes place so quickly and successively as is sometimes supposed; *time* is evidently required to effect the necessary changes. The subject, however, receives additional interest at the present time, inasmuch as the question of the application of the principle of *Conservation of Force* to physiological phenomena is now being brought prominently forward. These facts go far to prove that the principle may be safely applied to physiological phenomena, and at the same time also show the inconsistency, if not absurdity, of regarding the animal machine as nothing more than a mere thermo- or electro-dynamic apparatus.

I will briefly recapitulate, in a general manner, some of the most important conclusions that have been established in the foregoing inquiry in regard to the muscular power of the frog.

First. Out of thirty frogs taken indiscriminately during the latter end of the month of March and the beginning of April, 1864, 1 gr. weight of muscle could raise, in round numbers, 445 grs. through the space of $\frac{1}{5}\frac{1}{3}$rd of an inch; whereas during the summer months (the latter end of June and the beginning of July, 1863), the power of 1 gr. of muscle was equivalent to or could raise 608 grs. through the same space.

Secondly. That during the summer months the power of the male was equivalent to 1 gr. raising 656 grs., and in the female 1 gr. could raise 579 grs. But during the spring months, in the female 1 gr. could raise 484 grs., whilst in the male 1 gr. could raise only 431 grs.

Thirdly. That the sexual function has an important influence over the muscular power, for in those males that were copulating up to the time of experiment 1 gr. could raise 413 grs., whereas in those that were not copulating 1 gr. could raise 448 grs.

Fourthly. That during muscular action an increase in weight and vascularity occurs in the muscles.

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EXPLANATION OF THE PLATES.

Plate I.

To illustrate Dr. Beale's observations on very fine Nerve Fibres, &c., page 19.

The drawing represents a large plexus composed entirely of very fine fibres (under 1-60·000th of an inch in diamenter) from the bladder of the common frog. The trunks which enter into its formation are also composed of very fine fibres alone. Bundles of fibres are seen to cross from one side of the plexus towards the opposite. Indeed, the general arrangement of the very fine fibres composing this plexus is much the same as that observed in the case of the bundles of large nerve fibres which enter into the formation of the ordinary plexuses.

Nuclei are seen in connection with some of the fibres. To the left, a compound fibre consisting of eight or nine fine fibres is seen to pass off. Some of the fibres entering into the formation of this branch are derived from the upper, and others from the lower, part of the plexus. A similar arrangement is noticed in the case of all the other trunks. Indeed, all nerve trunks are composed of fibres which have proceeded from opposite directions.

The plexus delineated was not visible in the recent specimen, but after the prolonged action of dilute acetic acid, it, with many fibres composed of similar fine fibres, became perfectly distinct. Magnified 700 diameters.

Plate II.

To illustrate Mr. Lockhart Clarke's observations on the Structure of the Spinal Cord in a Case of Wasting Palsy, page 3.

Fig. 1.—Patch of degeneration from the lower part of the conus medullaris of the spinal cord. A portion of degenerate nerve fibres; *a*, the same becoming broken up or granular toward the edge of a transparent area B, in the centre of which is a group of the finest granules. C, degenerate nerve fibres at the opposite edge of the transparent area. × 420 (see § 1).

Fig. 2.—Transverse section of the lower part of the conus medullaris. *c, c,* posterior white columns; *e e,* anterior, white columns; *b,* posterior grey substance; *d,* anterior grey substance; *a, a,* morbid mass obliterating all the central parts of the grey substance, and containing small transparent patches represented at B, Fig 1; *s,* anterior median fissure. (See § 2.)

Fig. 3.—A transverse section from the same region of a healthy cord, showing the normal quantity and shape of the grey substance.

Fig. 4.—A transverse section through a higher part of the conus medullaris of the diseased cord. *b,* the transparent gelatinous substance; *a,* the morbid mass, replacing all the central parts of the grey substance, as seen on comparing it with.

Fig. 5.—Which represents a similar section (see § 3) of a healthy cord.

Fig. 6.—Transverse section of the diseased cord through the lower part of lumbar enlargement. (See § 4.)

Plate III.

To illustrate Mr. Lockhart's Clarke's observations on the Structure of the Spinal Cord in a Case of Wasting Palsy, page 31.

Fig. 7.—A transverse section of a healthy cord from the same as Fig. 6. *g,* posterior median fissure; *i,* anterior nerve roots; *f,* posterior nerve roots.

Fig. 8.—Transverse section through the upper third of the lumbar enlargement of the diseased cord. *c,* superficial portion of the white posterior columns, very much softened on the opposite side; *c′,* deeper portion of the same columns, affected by granular degeneration at the point where they rest on the transverse commissure of the grey substance; *k,* remains of the posterior vesicular column, from which a process of connective tissue separates the posterior white columns into two parts, *c, c′.* (See § 5.)

Fig. 9.—Transverse section of the grey substance of diseased cord at about the middle of the upper third of the lumbar enlargement. *c, c′,* posterior white columns; *k, k,* posterior vesicular columns; *e, e,* anterior white columns. (See § 5.)

Fig. 10.—Represents another section from the same cord, a little higher up. The form of the grey substance on each side is considerably altered. *m,* a pyriform patch of finely-granular degeneration, in the substance of the cervix cornu, on the

outer side of posterior vesicular column *k*. On the opposite side of the cervix cornu, *n*, is exceedingly narrow, and contains no traces of posterior vesicular column, but is joined to its fellow on the right side by processes of connective tissue, which divide the posterior white columns *c c'* into several irregular parts. (See § 5.)

Fig. 11.—A transverse section of the grey substance at the upper end of the lumbar enlargement, showing a curious deviation from the normal shape, as may be seen by comparing it with Fig. 12. (See § 5.)

Fig. 12.—Represents a corresponding section of one side, and part of the other from a healthy cord.

Plate IV.

To illustrate Mr. Lockhart Clarke's observations on the Structure of the Cord in a case of Wasting Palsy, page 36.

Fig. 13.—Transverse section of the grey substance of the diseased cord at the lowest part of the dorsal region. (See § 6).

Fig. 14.—A corresponding section from a healthy cord. Here the lateral halves, with their posterior vesicular columns, *k, k,* stand widely apart, and are joined only by the posterior transverse commissure. In the diseased section, fig. 13, *k k* are brought closely together; the form of the entire grey substance resembles that of the higher mammalia.

Fig. 15.—A transverse section of the grey substance of the diseased cord at the upper part of the lower third of the dorsal region. Behind *c* the posterior cornua are united by a process *a* of connective tissue; and between *c* and *c'*, the two remaining portions of the posterior white columns in the central line, were several morbid patches, *b, b, b,* of transparent granular degeneration. The whole form of the grey substance is strangely altered. (See § 6).

Fig. 16.—A transverse section of the grey substance of the diseased cord at the lower part of the middle third of the dorsal region. Its two lateral halves are unsymmetrical. Compare *d, d, k, k,* and *t, t.* (See § 6).

Fig. 17.—A transverse section of the grey substance of the diseased cord at the lower part of the upper third of the dorsal region. *k, k,* Posterior vesicular columns; *t, t,* tractus intermedio-laterales; *b, b,* several clots of blood, effused from a varicose and degenerate vessel, in the centre of the right lateral half of the grey substance. (See § 7).

Fig. 18.—Part of a similar section about two lines higher up, magnified 60 diameters. *p*, The central canal of the cord, surrounded by epithelium; *s*, the anterior median fissure; *k*, the posterior vesicular column of the right side; *t*, the tractus-intermedio-lateralis of the same side. Immediately in front of *k*, and extending into *t*, was a large irregular clot of blood, *b*, *b*, *b*, imbedded, as in fig. 17, in the central portion of the right lateral half of the grey substance. (See § 7.)

Plate V.

To illustrate Mr. Lockhart Clarke's observations on the Structure of the Cord in a case of Wasting Palsy, page 37.

Fig. 19.—A transverse section of the entire grey substance of the diseased cord, near the middle of the dorsal region. The anterior cornu, *d*, on the left side, is much smaller than on the right, and is thrown or drawn backward away from the anterior median fissure, *s*, together with the corresponding tractus intermedio-lateralis, *t*, the posterior cornu, and its posterior vesicular column, *k*. (See § 7.)

Fig. 20.—A transverse section of the grey substance of the diseased cord at the lower part of the cervical enlargement. Posterior horns cut off for economy of space. *b*, *b*, Four dilated blood-vessels cut more or less transversely, in the centre of the grey substance around the canal. (See § 8.)

Fig. 21.—A similar section a few lines higher up. In the centre of the grey substance, the cut ends of two dilated blood-vessels are seen to be surrounded by a finely granular area of degeneration, *b*, which destroyed the posterior commissure. (See § 8.)

Fig. 22.—A similar section about half an inch higher up. The morbid area, *b*, triangular in form, has increased in extent, and encroached backward on the under part of the posterior columns, *c'*, which, at this spot, are also affected by granular degeneration. (See § 8.)

Figs. 23 & 24.—Two similar sections nearer the middle of the cervical enlargement. *b*, *b*, Show the form and extent of the morbid area. (See § 8.)

Fig. 25.—A similar section at the middle roots of the third dorsal nerves. (See § 8.)

Fig. 26.—Another section a few lines higher up. *a*, Granular remains of the anterior commissure, surrounded by *b*, *b'*, a transparent area of degeneration. (See § 8.)

Plate VI.

To illustrate Mr. Lockhart Clarke's Observations on the Structure of the Cord in a case of Wasting Palsy, and Dr. Beale's Observations on Mucous Casts from the Uriniferous Tubes, pages 39, 40, 56.

Fig. 27.—A similar section opposite the middle roots of the second dorsal nerves. The morbid area, *b*, *b*, after having diminished gradually, and nearly disappeared, again increases in extent. At *b*, the posterior commissure, and parts surrounding the canal, were quite broken up, consisting of bits of nerve fibres, of granules, and of numerous corpora amylacea. (See § 9.)

Fig. 28.—A similar section at the upper roots of the second dorsal nerves. The transparent patches of degeneration are prolonged backward on the right side, as well as on the left; and for a few lines upward along the cord, the whole inner portions of the cervix cornu were destroyed by irregular patches of granular degeneration, *b*, *b′*, *b″*, *b‴*, like a piece of moth-eaten cloth. At *s*, an enlarged and unhealthy-looking blood-vessel is seen entering through the anterior median fissure, and crossing the lower area of degeneration. (See § 9.) This sketch has been shaded to represent the grey substance, and show the morbid spaces more distinctly. (See § 9.)

Fig. 29.—A similar section opposite the lower roots of the first dorsal nerves. The morbid area, *b′*, *b″*, acquires its largest dimensions. (See § 10.)

Fig. 30.—Large nerve-cells, variously affected by atrophy and degeneration. (See § 11.)

Fig. 31.—Casts composed of transparent mucus from the straight portion of the uriniferous tubes from a case of renal irritation. Magnified 150. (See page 56.)

Plate VII.

To illustrate Dr. Marston's Paper upon Aneurism of the Aorta, communicating with the Pulmonary Artery, page 63.

Fig. 1.—Loud bronchial and crepitant râles heard over these parts. 2. Region of hepatic dulness. 3. Dull from the presence of fluid (Ascites). 4. Resonant. 5. Centre of cardiac dulness, and seat of purring tremor. 6. Apex beat to the left of nipple; seat of a double murmur (the diastolic one

being faint). 7. Double murmurs of maximum intensity (the diastolic being the loudest) heard in the direction of the arrows. 8. Seat of an undulating impulse of the right heart.

In the area marked out by the figures 6, 7, 8, cardiac murmurs were audible.

Fig. 2.—6 marks the situation and extent of splenic disease.

Plate VIII.

To illustrate Dr. Ogle's Paper on Laceration of the Lower part of the Right Internal Jugular Vein, caused by Fractured Portions of Clavicle, page 125.

The lacerated opening in the internal jugular vein, is seen at *a*.

Plate IX.

To illustrate Dr. Beale's Paper upon the Branching of Nerve Trunks, and the Sub-division of the Individual Fibres composing them, page 127.

Fig. 1.—The course of the branches in a large compound nerve-trunk, or of the fine fibres in a very fine nerve-trunk, at the point where a branch is given off at right angles.

Fig. 2.—The course and division of individual dark bordered nerve-fibres, showing that one of the fibres, resulting from the sub-division of a fibre, passes on in the direction of the original fibre, while the other passes into the branch which diverges from the trunk. *a* and *d* may be commissural fibres between two sets of cells at the periphery, or between two sets of central cells. The arrows indicate the direction which the nerve-currents in the different fibres may take.

Fig. 3.—Branching of a fine nerve-trunk, from the pectoral muscle of the frog, showing the course of the respective dark-bordered fibres in the trunks, and their division.

Fig. 4.—A fine trunk composed of very fine nerve-fibres from the mylo-hyoid muscle of the hyla viridis, magnified 1,800 diameters. The general arrangement in the case of these very fine fibres, is the same as that observed in dark-bordered nerve-fibres, and in compound nerve trunks.

Fig. 5.—Diagram to illustrate the manner in which the nerve-fibres distributed to the papillæ of the tongue of the frog are arranged. It will be observed that the papillæ are con-

nected to one another by commissural fibres, while some of the fibres of the nerve-trunk are not connected with all the papillæ figured. There are also fibres in the trunk which are not distributed to any one of the three papillæ in the figure. The nerves end in a net-work or plexus, at the summit of each papillæ.

Fig. 6.—A nerve dividing into three branches. From the breast muscle of the frog. At *a* and *b*, individual dark bordered fibres are seen to divide, the fibres resulting from the sub-division passing onwards in different branches. See fig. 2.

Fig. 7.—Fine trunks resulting from the sub-division of a small branch of the nerve distributed to the breast muscle of the frog.

Plate X.

To illustrate Mr. Lockhart Clarke's Pathological Observations in a Case of Epilepsy, accompanied by Diabetes, page 146.

Fig. 1.—Substances found in a rust-coloured mass about the size of a hazel-nut, near the middle of the inner third of the right optic thalamus.

a. Oblique rhombic prism of hematoidin. *b*. Three crystals of the same embedded in mass of fat globules. *c, d, e, f*. Amorphous granules of hematoidin interspersed through oil globules of different sizes. *b, f, g', h*. Oil globules, stained of a yellow or brown colour, by a solution of hematoidin. *j, l*. Exudation corpuscles, consisting of minute globules of fatty matter. *k*. Acicular and radiating crystals of margarine.

Fig 2.—A transverse section of the medulla oblongata on each side of the median line, a little above the point of the calamus scriptorius.

f, e, f'. The median portion of the floor of the fourth ventricle.

a. The posterior pyramid of the left side, forming the outer part of the floor.

b, b'. The vesicular nucleus of the uppermost roots of the spinal-accessory nerve of the same side; immediately below *b'* is a dark oval mass consisting of bundles of longitudinal fibres. The posterior surface of this nucleus with that of its fellow of the opposite side, here forms the point of the calamus scriptorius, and the floor of the fourth ventricle, *f, e, f'*, on each side of the medial line.

c. The space surrounding the elongated canal.

d. The vesicular nucleus or centre of origin of the lingual

nerve of the left side; on the opposite side its large and variously shaped cells are represented.

g, *g'*. The roots of the lingual nerves.

h. The raphè.

At this level of the medulla oblongata, the surfaces of the spinal-accessory nuclei *f*, *f'*, forming the median floor of the fourth ventricle, were covered with a layer of oil-globules, resulting evidently from degeneration of their superficial portions, which were ragged, uneven, and, as it were, eroded. In the median furrow also their natural substance was completely destroyed, and replaced by a deep column of densely-crowded oil-globules, *e*, which were continuous with those on each side *f*, *f*. The point of the calamus scriptorius was composed entirely of this fatty column; and a similar degeneration, but in a less degree, extended to the corresponding parts a little below, as well as a little above, where it partly affected the nuclei of the pneumogastric nerves. All the remaining, deeper portions of the spinal-accessory nuclei *b*, *b'*, were scattered at intervals with isolated oil-globules of considerable size; and the space between them, *c*, surrounding the canal, contained them in still greater numbers. The posterior pyramids *a*, *a*, were likewise interspersed with the same kind of globules, which were to be found, also, more sparingly scattered even through the restiform bodies. The cells of the spinal-accessory nucleus were normal, many of them being filled, as usual, with dark brown, or nearly black pigment granules. The lingual nuclei contained no oil-globules, and their cells were normal in shape and size, but enclosed rather more pigment than usual.

Plate XI.

To illustrate Dr. Beale's Observations upon the Anatomical Controversy concerning the distribution of Nerves to Voluntary Muscles. *Page* 160.

Fig. 1.—Pale fibres with peculiar bodies (Endknospen), said to exist beneath the sarcolemma by Kühne. This is a part of Kühne's fig. 7, Plate II., accompanying his memoir "Uber die peripherischen Endorgane der motorischen Nerven." The white substance is said to cease at the sarcolemma, while the axis cylinder is continued beneath as an intramuscular pale fibre with nervenendknospen.

Fig. 2.—A part of one of Kölliker's figures, copied from the Croonian Lecture (May, 1862), published in the Proceedings of

the Royal Society, vol. xii., p. 60. "Termination of a dark-bordered tubular nerve-fibre on a muscular fibre *f*, of the cutaneous muscle of the frog, seen with Hartnach's objective à immersion No. 10, and eye-piece No. 1. *a*, sheath of the nerve fibre continued upon the pale terminal fibres; *c c*, nuclei of the pale fibres; *d*, a nucleus belonging to the muscular fibre *f*, *e e e*, ends of the pale fibres; *b*, nucleus of the dark-bordered fibres. The figure shows the continuation of the dark-bordered fibres into the pale filaments, but fails to exhibit the faint outline and characteristic aspect of the latter."

The fine fibres which Kühne maintains lie beneath the sarcolemma and are intra-muscular, are believed by Kölliker and myself to lie upon the external surface of this membrane, and to be inter-muscular.

Fig. 3.—Division and subdivision of fine dark-bordered fibres on the surface of muscular fibre, showing numerous fine fibres arranged in the form of a network, and nuclei connected with these fine fibres. Two or three of the delicate compound nerve fibres may be traced over the edge of the muscular fibre. Pectoral muscle of the frog, 700.

This figure was copied by myself from a muscular fibre from the very same muscle of the frog (pectoral) as that from which the figures of Kühne and Kölliker were obtained. I have copied exactly what I saw. That the fine and pale, as well as the coarse fibres, are inter-muscular and lie upon the external surface of the sarcolemma, is quite certain, as they may be traced over the edge of an elementary fibre, and may be followed from one to the surface of another.

The fibres considered by Kühne and Kölliker to be single pale fibres really consist of several very delicate fibres, and the appearance which has been regarded as the outline of the sheath, is in many cases caused by the presence of very fine fibres, which mingle with the fine fibres which form the direct continuation of the dark-bordered fibres. By comparing these three figures with each other, it will be observed that the constitution of the "pale terminal fibres" is represented very differently. Much of what Kölliker has figured really seems to constitute the sheath, and the lines which, according to him, represent the outline of the sheath, are, according to me, fine nerve fibres. He and Kühne consider that the fibres really end where they are made to cease abruptly in their drawings. On the contrary, what seem to be ends in my drawings merely represent the point beyond which I could not follow the fibre in the particular specimen figured, in consequence of its ex-

treme transparency. I am confident the fine fibres do not end at all, but that these very fine compound but scarcely visible nerve-fibres pass to other trunks, and thus a network is formed with lax meshes. This network is much obscured by the intermuscular connective tissue, but it lies upon the elementary muscular fibres, just external to the sarcolemma. This network of nerve fibres has not yet been observed in this or other tissues by Continental observers, and is still included in the "connective tissue."

Figs. 4, 5, 6, and 7, are copied from Kühne's paper in Virchow's Archiv., vol. 27, Plate XI. In this paper the intramuscular pale fibres for the most part lose their definite character, and their peculiar nerve 'knospen'—represented in the first paper. The axis cylinder is now lost in a collection of granular matter and nuclei, arranged in the most irregular manner.

Fig. 4.—From the psoas of a rabbit. *a.* Nucleus of the nerve sheath. *N.* Nerve. *H.* Nerve prominence (Nervenhügel). *b.* Nuclei in the nerve eminence. *c.* Muscle nuclei. *d.* Glittering globules in the granular contents of the nerve eminence.

Fig. 5.—From one of the muscles of the eye of a dog. The explanation of the letters as above, *a'*, a nucleus with granular contents × 450.

Fig. 6.—From the gastrocnemius of the rabbit. Explanation as above × 450.

Fig. 7.—Also from the gastrocnemius of the rabbit. The nerve eminence somewhat pushed on one side by the pressure of the covering glass × 450. The explanation as in fig. 5.

[I am of opinion that the nerves and nerve nuclei all lie external to the sarcolemma, not beneath it as represented in these drawings. By Kühne's mode of preparation, the nuclei have been completely dislocated from their original orderly mode of arrangement, and the nerve fibres passing between them have been destroyed. The nuclei of the nerve sheath, marked *a*, are, I maintain, in many instances, the nuclei of fine nerve fibres running in the same sheath with the dark-bordered fibres.]

Fig. 8 is copied from a part of another drawing of Kühne's, published in Virchow's Archiv., vol. 28. It represents the nerves in what Kölliker has termed 'nodular swellings,' or nerve tufts, in the breast muscle of the frog. These bodies contain very young muscular fibres, which gradually increase in size, and thus the width of the muscle is from time to time increased. In this figure the dark-bordered fibre is represented as ter-

minating abruptly in a granular nucleated mass. Neither pale fibres, nervenendknospen, nor nervenhügel, are here represented. They may not be developed yet. But the huge dark-bordered fibre is made to end apparently by a wide mouth, which opens into a space beneath the sarcolemma. The terminal nerve fibre is nearly as wide as the tube of the sarcolemma itself, at the point where it becomes intramuscular!

[The further narrowing of these dark-bordered fibres, and their division into finer branches, which run for a long distance upon the surface of the sarcolemma, beyond the point where Kühne makes them enter, cannot be seen in specimens prepared according to the plan followed by this observer. It is quite certain that dark-bordered fibres do not terminate in any tissue in the manner suggested by this figure. I have seen a complete leash of very fine nerve fibres moving over fine muscular fibres like those represented in this figure.]

Fig. 9.—Portion of an elementary muscular fibre from one of the abdominal muscles of the white mouse, with four dark-bordered fibres (*a*) crossing over its surface; a capillary vessel (*b*) is also seen with fine nerve fibres distributed to it. A few of the transverse markings of the muscle are represented at the left-hand corner of the fibre. Two of the dark-bordered nerve fibres (*c*) pass over the elementary fibre, to be distributed to adjacent fibres. One dark-bordered fibre (over *d*) is lost among several pale nucleated fibres. Pale nucleated fibres are seen ramifying in several places upon the surface of the elementary fibre, but beneath the dark-bordered nerve fibres and capillaries. It will be observed that a fine fibre, which seems at the upper part of the figure to be but one of the outlines of the tubular membrane of one of the dark-bordered fibres, leaves the dark-bordered fibre and passes to the capillary vessel (over *e*). I have so often observed a dark-bordered nerve fibre distributed to muscle, divide into two branches, one of which passed to a vessel, while the other ramified upon the muscle, that I believe the arrangement to be usual, and probably essential.

The capillaries, dark-bordered nerve fibres, and pale nucleated fibres, represented in this drawing, as well as the intervening connective tissue, can be completely stripped off from the surface of the sarcolemma, without tearing that membrane. Of the structures, therefore, represented in this drawing, not one can lie beneath the sarcolemma.

Fig. 10.—A small portion of fig. 9, over letter *f*, much more highly magnified. A portion of the capillary vessel is

marked *a*. One of the dark-bordered nerve fibres *b* is seen over *c* to lose itself amongst the number of pale nucleated fibres. The numerous nuclei in this small space are of the nature of those represented by Kühne as lying beneath the sarcolemma, but as this observer has not injected the capillaries with preservative fluid immediately after the death of the animal, the delicate structure here represented has undergone disintegration, and therefore could not be seen by him.

Figs. 9 and 10 I offer as an explanation of the appearances represented by Kühne in figs. 4, 5, 6, 7, and I think that the nuclei irregularly arranged amongst granular matter and placed beneath the sarcolemma in his drawings, are the nuclei connected with the pale fibres outside the sarcolemma in my fig. 10. It is clear that we are at issue upon a very broad question of fact. The very same nuclei which, according to the observations of Kölliker and myself, lie outside the sarcolemma, are placed by Kühne beneath this membrane.

Plate XII.

Illustrating Dr. Beale's Observations upon the Anatomical Controversy relative to the distribution of Nerves to Voluntary Muscles. *Page* 160.

Distribution of finest nucleated nerve-fibres to the very narrow elementary muscular fibres of the mylo-hyoid of the little green tree-frog (Hyla arborea), magnified 1800 diameters.

The elementary muscular fibres are marked *g, h, i, k*. *k* is a very young one, slightly stretched; *i* is a fully formed muscular fibre; *h*, another stretched in its central part. The nuclei of these fibres exhibit some differences in size and form. Nucleoli are distinct in all, and in the fibre marked *g* the nuclei, which were coloured by carmine, exhibit three different intensities of colour—the dark central spot, "nucleolus," being most intensely coloured, as indicated by the shading in the drawing.

a is a nerve fibre which was followed over more than twenty elementary muscular fibres from a dark-bordered fibre. One of the subdivisions of this fibre is seen at *f*, where it again runs with a very fine dark-bordered fibre (*o*). The dark-bordered fibre (*o*) was some distance higher up in the specimen, but its place has been altered in order to avoid the necessity for a still larger drawing. Above *b* a nucleus of a very fine nerve fibre is seen. Such nuclei lie upon the surface of the muscular fibres, external to the sarcolemma. The nucleus often appears as

if it were within the sarcolemma (*c*), but the fibres proceeding from each extremity render such a position impossible. The relation of these nerve-nuclei to the sarcolemma is seen at *l* in profile. The nuclei, as well as the fibres for a certain distance, often adhere to the sarcolemma very firmly; but in the thin mylo-hyoid muscle the course of the fibres over or under, but always *external*, to the muscular fibres, may be readily traced if the muscular fibres be separated slightly from each other, as represented in the drawing.

At *d* fine nerve-fibres accompanying the fine fibre continued from the dark-bordered fibre, as described in the "Philosophical Transactions" for 1862, are represented. Such fibres are also seen at *e* and *f*.

m, *n*, and *o*. Dark-bordered fibres, with nuclei near their distribution. *m* Would probably pass over sixty or seventy muscular fibres, and *n* over perhaps twenty, before it divided into fibres as fine as those seen at *b*, *e*, *f*, *l*.

p. A very fine capillary vessel with a nerve-fibre running close to it.

q. A bundle composed of six very fine nerve-fibres near their distribution. These fibres exhibit a very distinctly beaded appearance, which is also observed in many other fine fibres in different parts of the specimen.

Traces of connective tissue are seen in all parts near the fine nerve-fibres and around the muscular fibres. Here and there some very fine connective tissue-fibres, which were not altered by acetic acid, are represented. These represent the remains of fine nerve-fibres, which existed in a state of functional activity at an earlier period.

The drawing, with the exception of the position of the nerve-fibre (*o*) above mentioned, is an actual copy from nature. The relative position of the muscular fibres, the form and general character of the so-called nuclei, and the position and size of the nerve-fibres and their nuclei, have been carefully preserved.

The very fine nerve-fibres have been traced, in so many instances, from one trunk to another ramifying at a very considerable distance, that it is almost certain that no true terminations or ends exist.

Plate XIII.

To illustrate Dr. Beale's observations upon the controversy relating to the Distribution of Nerves to Striped Muscle, page 160.

Fig. 1.—Dark-bordered fibres. The nucleated dark-bordered fibre *a* is seen to divide into two branches. The one

passing in the division which diverges to the right, rapidly becomes so very fine that it can hardly be followed as it runs parallel with the other dark-bordered fibre and in the same sheath with it (under *d*). This very fine fibre, in direct continuity with the dark-bordered fibre, cannot be distinguished from the fine fibres usually regarded as connective tissue seen in the sheath of the nerve on the left side of the specimen (at *b*), which leave the trunk and assist in forming a separate branch at *c*. From the pectoral. × 700. The finest nerve-fibres are really much finer than is generally supposed.

Fig. 2.—Dark-bordered nerve-fibre with fine fibres on one side of it. These fine fibres appear in one part like the outline of the tubular membrane, and are usually considered to be connective tissue, but they are seen to give off branches which, with other fine fibres of the same character, form networks.

Fig. 3.—Fine fibres from the sheath of the nerve, dividing and subdividing on the surface of two elementary muscular fibres. From muscle of the neck of the frog. × 700 diameters. Compare the appearance of the fibres represented at *b*, *b* in the drawing, with fibres *b* in figs. 1 and 5, which undoubtedly come from true dark-bordered nerve-fibres.

Fig. 4.—Very fine nerve-fibres on the surface of the muscular fibre. From the pectoral of a young frog. Several of these fibres can be followed to the edge of the muscular fibre, where they are lost. × 1800.

Fig. 5.—Young muscular fibre from the pectoral muscle of the frog, near one of the oval swellings. At *a* and *b* are seen the fibres which are continuous with the dark-bordered fibres. These are gradually lost amongst very fine fibres. The compound branches divide and subdivide on the surface of the muscular fibre. × 700.

Fig. 6.—Fine dark-bordered fibre, and fine nerve-fibres which are generally looked upon as fibres of connective tissue in the sheath of the nerve, with nuclei. From muscle of the neck of the frog. × 700.

Plate XIV.

To illustrate Dr. Beale's observations upon the controversy relating to the Distribution of Nerves to Voluntary Muscle, page 160.

Fig. 7.—Division of dark-bordered fibre with fine fibres ramifying in the sheath of the nerve. From the pectoral muscle of the frog. × 700. The fine fibres, it will be

observed, break up into bundles composed of still finer fibres. These can be followed for some distance, and at last, from their extreme tenuity, they are lost.

Fig. 8.—Division of dark-bordered fibre on surface of muscular fibre. Pectoral muscle of the frog. The fibres resulting from the subdivision of the dark-bordered fibre may be traced for some distance amongst the very fine fibres.

Fig. 9.—Division of dark-bordered fibres and formation of networks of pale compound and very fine fibres. Also from the pectoral of the frog.

Fig. 10.—Branching and division of dark-bordered nerve-fibre near terminal branches, and bundles of fine fibres. Several nuclei are seen in connection with both sets of fibres. From the bladder of the frog. × 700. At *a* the dark-bordered fibre becomes resolved into several fine fibres. It is in this way that the dark-bordered nerve-fibre invariably 'ceases.' It is not the "axis cylinder" which is alone continued, as is usually stated, but the substance of the fibre breaks up to form a bundle of very delicate fibres. This bundle divides and subdivides, and, with bundles from other fibres, helps to form a network.

Plate XV.

To illustrate Dr. Beale's observations upon the controversy relating to the Distribution of Nerves to Striped Muscle, page 160.

Fig. 1.—Eight muscular fibres seen in section, showing the manner in which, according to Kühne, the nerve-fibre perforates the sarcolemma, and comes into close relation with the contractile tissue. M., Muscular fibres; N., nerve-fibres; *b*, collection of nuclei of the nerven-hügel.

Fig. 2.—Part of another drawing more highly magnified, to illustrate the same point. Also after Kühne.

Fig. 3.—Copy of a schematic figure reduced one-half. After Kühne. This figure illustrates the latest inferences of this author upon the termination of motor nerves in the muscular fibres of amphibia and mammalia, including man. A, axis cylinder and its expansion as an end-plate; *a a*, white substance; *b*, sheath; *c*, nuclei of the sheath; *d*, a nucleus of the same kind in the membrane of the nerven-hügel; B, contents of the nerve eminence or nerven-hügel, a granular substance (Protoptasm?); C, the clear nuclei of the nerve eminence, with their large nucleoli; D, solitary large

nucleoli (?) in the granular contents of the nerve eminence; E, nucleus of the muscle; F, contractile tissue with the sarcous elements; G, sarcolemma.

Fig. 4.—Muscular fibre from the maggot. (L. Beale.) The sarcolemma is removed from the upper part. A bundle of nerve-fibres is seen crossing the muscle, from which finer but still compound fibres proceed. To the right a branch may be traced downwards to the sarcolemma, which is drawn out so as to form a little eminence (nerven-hügel) at the point where the nerve comes into contact with it. The nerve-fibre divides at this point into finer fibres, which ramify upon the surface of the sarcolemma, and are adherent to it. Above and below the nerve-fibre are numerous branches of tracheæ, which also ramify upon the sarcolemma, and are intimately adherent to it.

Fig. 5.—Represents one of these so-called nerve eminences more highly magnified. Immediately within the fold of the sarcolemma is one of the large nuclei of the muscle. The nerves forming the fine nerve-trunk (*b*) pass, some on the anterior, and some on the posterior, surface of the sarcolemma, as represented in the drawing. *a, a,* are branches of tracheæ.

Plate XVI.

To illustrate Dr. Beale's observations relating to the Distribution of Nerves upon Striped Muscle, page 160.

Fig. 1.—A nerve-tuft or swelling. After Kölliker. This is described as "a nodule or swelling of a muscular fibre (or rather fasciculus), with a large nerve-fibre, nerve-tuft, and nuclei; highly magnified."

Fig. 2.—A portion of one of the nerve-tufts or swellings from the breast muscle of the frog. Prepared according to the process alluded to in page 164. The dark-bordered fibres are seen to divide and subdivide into very fine fibres, which run amongst the developing muscular fibres which are distinctly seen in the drawing. The transverse markings can be discerned in many places.

At *a* is a portion of a young muscular fibre, to show the manner in which the contractile tissue is formed by the agency of the "nuclei," or masses of germinal matter.

Fig. 3.—From the same, more highly magnified. The muscular fibres and their nuclei are omitted, in order to show the subdivision and mode of ramification of the nerve-fibres more distinctly. The fine nerve-fibres ramifying in the sheath of the nerve are represented in figs. 2 and 3.

Plate XVII.

To illustrate Mr. Lockhart Clarke's Observations on a case of Paraplegia, p. 273.

Fig. 1. Sponge-like mass connecting the dura mater of the cord to the body of the eighth dorsal vertebra, under a low magnifying power. Part of it is of the nature of villous cancer.

Fig. 2. Isolated cells from texture represented in Fig. 1, × 420.

Fig. 3. Columns of cells from the same, with bundles of nucleated fibres separating them. The processes of the cells could be traced into these fibres.

Fig. 4. Nucleated cells, &c., from the cancerous growth. These are described on page 275.

Fig. 5. Pigment in a free state, external to the cells.

Fig. 6. Net-work of fine tough fibres, of which some hard yellowish masses embedded in the growth were composed.

Fig. 7. Cells and fibres composing the loops and villous processes of the cancerous growth.

Fig. 8. Two of the spaces in the interlacing and contorted bands.

Plate XVIII.

To illustrate Mr. Lockhart Clarke's Observations on a case of Paraplegia, p. 273.

Fig. 9. Remarkable structure, showing tubes (probably blood-vessels) surrounded by cancer cells.

Fig. 10. *q.* One of the processes connected with the cellular bodies represented at *p.*, Fig. 9, highly magnified. *r.* Darker and coarser bodies embedded in the tissue.

Fig. 11. Grey substance of spinal cord, much diseased and misshapen. *a, a, a.* Areas of transparent disintegration. *b.* Portion of left anterior cornu in a state of disintegration. *d.* Space at the bottom of the anterior median fissure which had undergone the same sort of change.

Fig. 12. Transverse section of the cord at the middle of the second dorsal nerve. For explanation, see page 281.

Plate XIX.

To illustrate Dr. Ogle's Observations on Neuromata, p. 322; *Mr. Kesteven's Remarks on a small Subcutaneous Tumour*; *and Dr. Tonge's Paper on the Development of Fungi in the Kidney*, p. 314.

Fig. 1. Shows the neuromatous tumour nodulated on its surface, and dissected from its attachments for the most part, and having the separated fibres of the nerve spreading out in a fan-like manner on its surface.

Fig. 2. Shows another aspect of the tumour, and displays the central hollow or cavity which it possesses. The tibia and fibula are represented.

Fig. 3. Elements seen in a thin section of a small subcutaneous tumour. Fibres of white and yellow fibrous tissue varying much in size; epithelial cells; crystals of cholesterine.

Fig. 4. Fungi from kidney.

Plate XX.

The figures in this plate illustrate the division and subdivision of bundles of nerve fibres to form networks or plexuses, p. 325.

Fig. 1. Networks of motor nerves on the mylo-hyoid muscle of the hyla, with branches of the artery and capillaries.

Fig. 2. Networks of sensitive nerve fibres from the eyelid of the frog.

PLEXUS OF VERY FINE NERVE FIBRES FROM THE BLADDER OF THE FROG

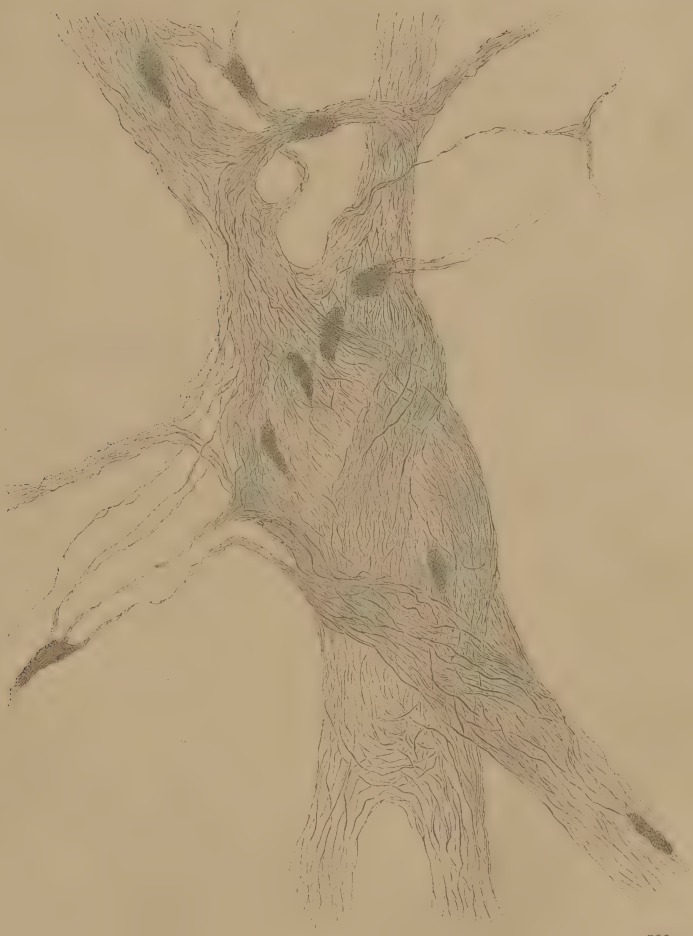

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Fig. 1.

Fig. 2.

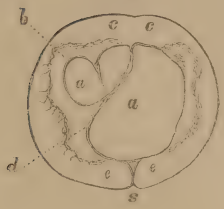
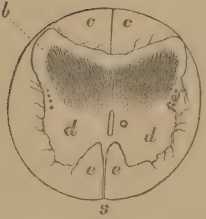


Fig. 3.



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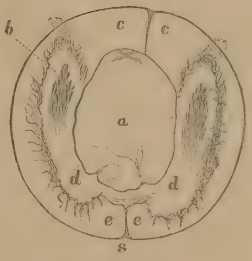

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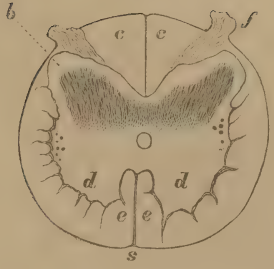


Fig. 6.

Fig. 7.

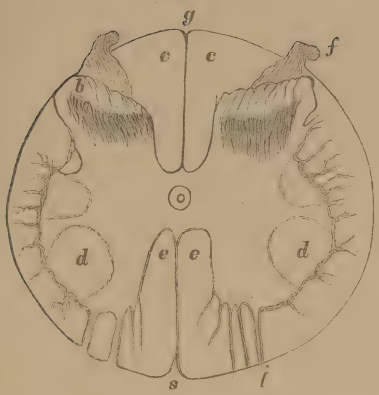
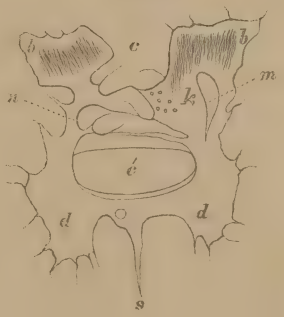


Fig. 8.

Fig. 9.

Fig. 10.



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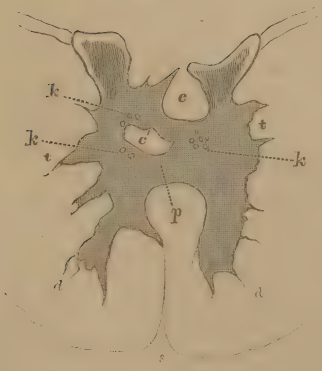


Fig. 12.

Fig. 13.

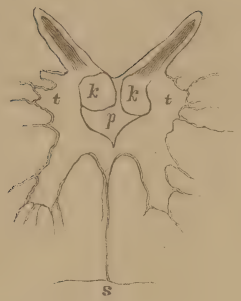


Fig. 14.

Fig. 15.

Fig. 16.

Fig. 17.

Fig. 18.

Fig. 19.

Fig. 20.

Fig. 21.

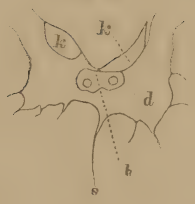


Fig. 22.

Fig. 23.

Fig. 24.

Fig. 25.

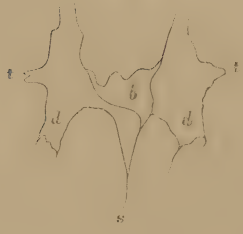


Fig. 26.

Fig. 27.

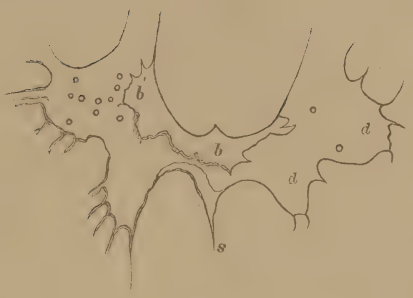
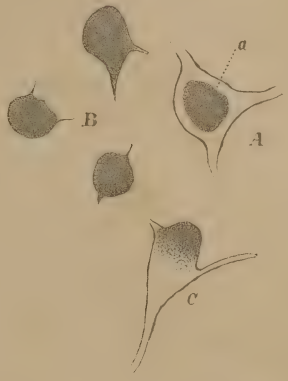


Fig. 28.

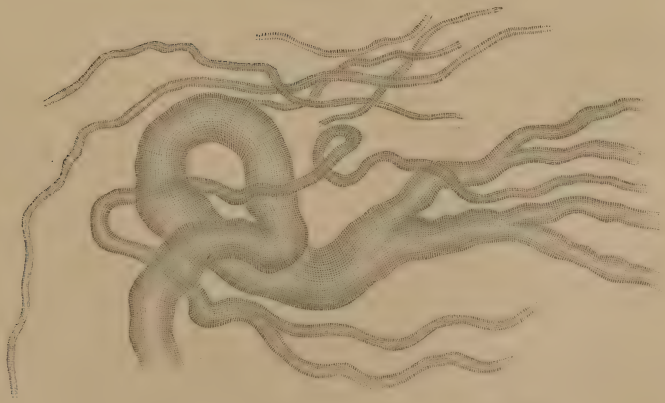
Fig. 29.

Fig. 30.



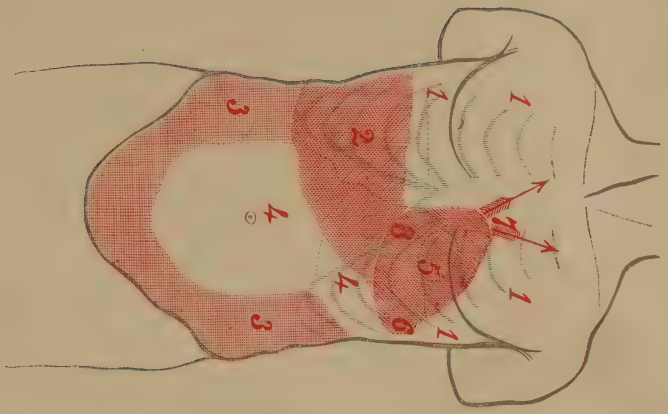
J. L. C. del.

Fig. 31.



× 150

J. S. B. del.


3
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1
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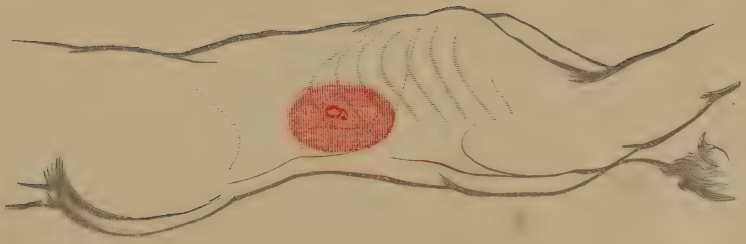
6
1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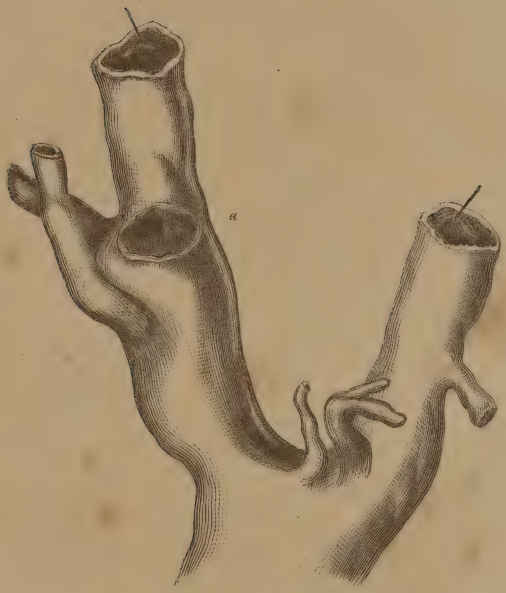
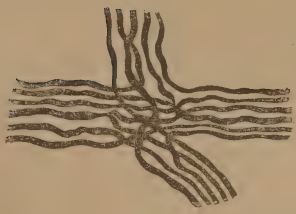


Internal jugular vein lacerated (*a*) by fractured portions of clavicle.—See Dr. Ogle's Paper, page 125.

Fig. 1.

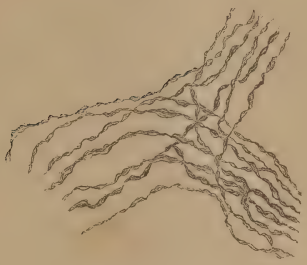
Fig. 2.

Fig. 3.



× 215.

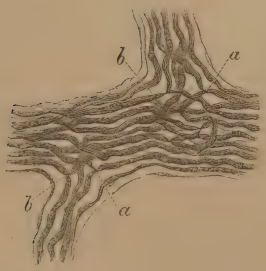
Fig. 4.



× 1800.

Fig. 5.

Fig. 6



× 21[illegible].

Fig. 7.

× 21[illegible].

Fig. 1.

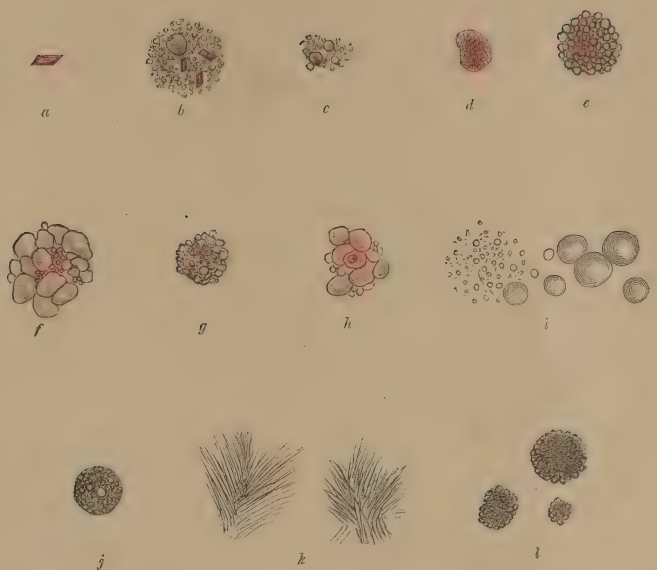


Fig. 2.

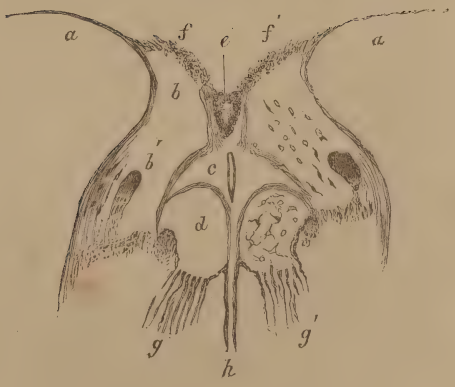
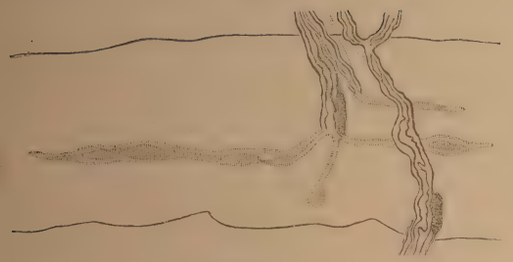
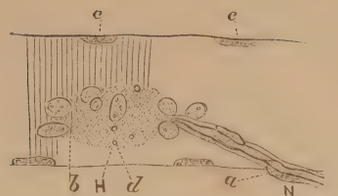


Fig. 1.



Kühne.

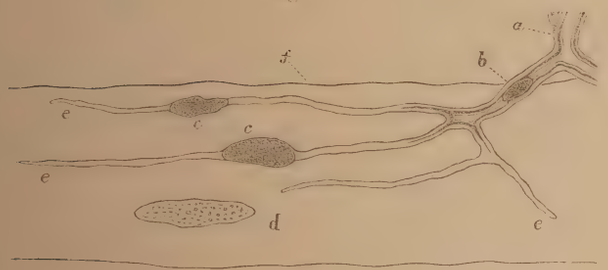
Fig. 4.



× 450.

Kühne.

Fig. 2.

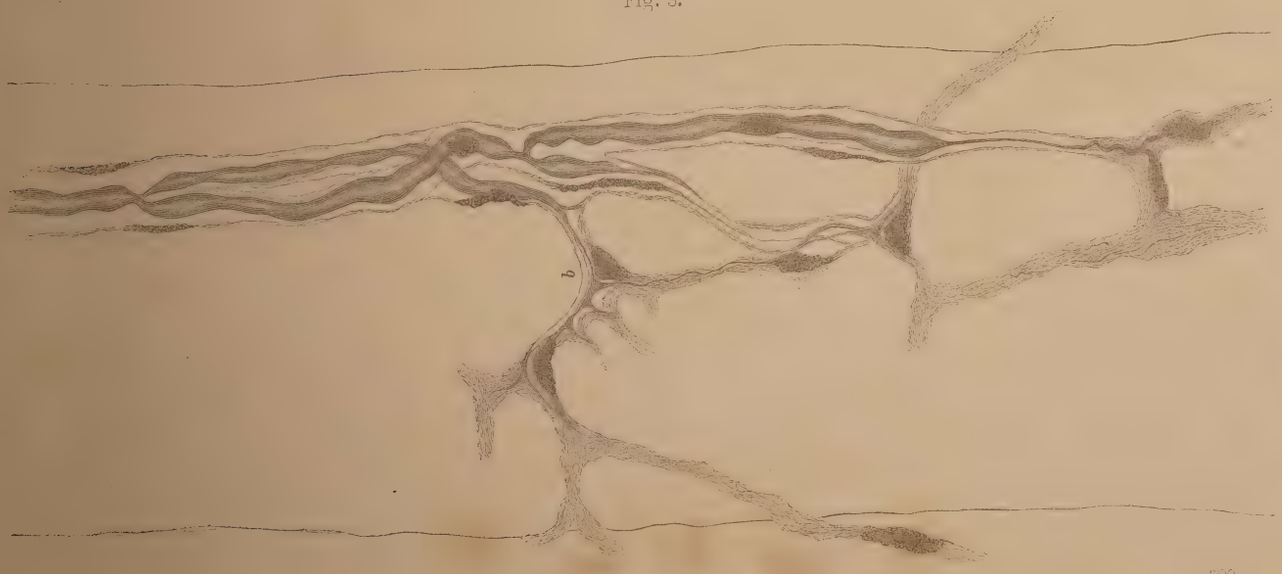


Kölliker.

Fig. 8.

Kühne.

Fig. 3.



Beale. × 700.

1000th of an inch × 700.

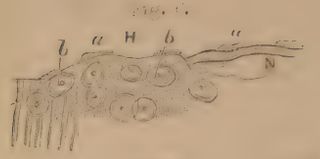
1000th of an inch × 180.

Fig. 6.

× 15

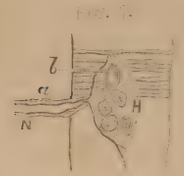
Kühne.

Fig. 7.



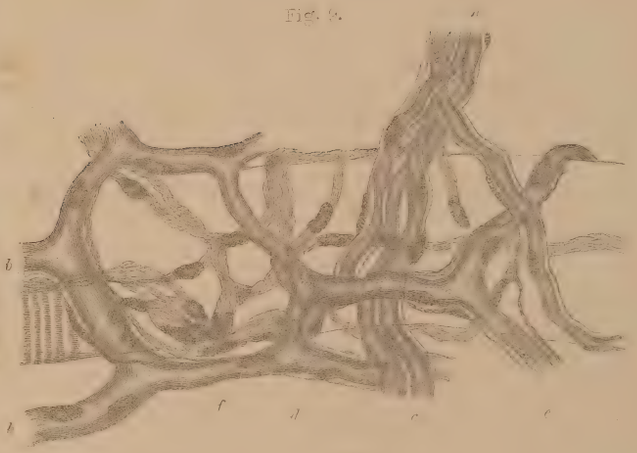
Kühne. ×

Fig. 5.



Kühne. ×

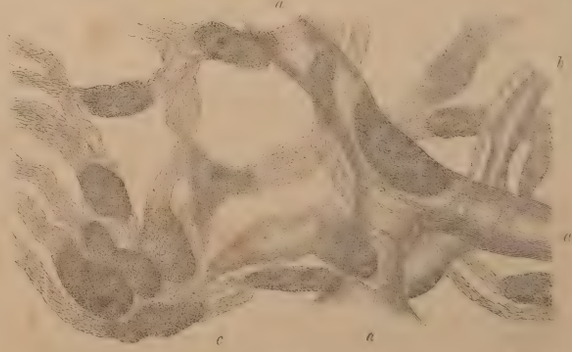
Fig. 9.



Beale

×

Fig. 10.

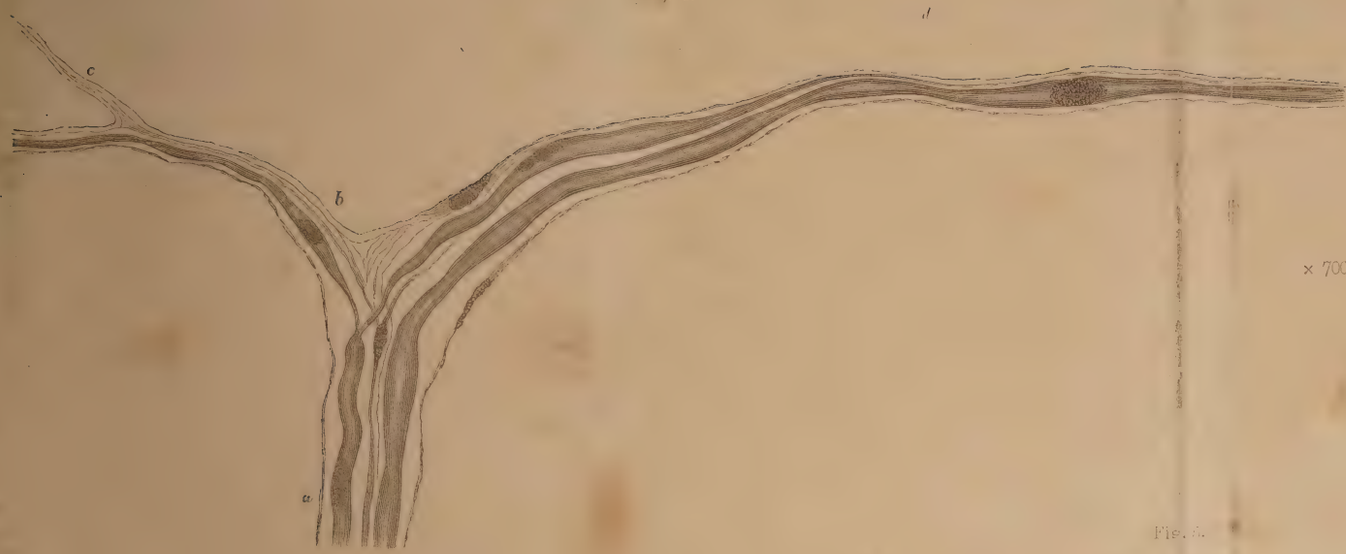


Beale. ×

Distribution of finest nucleated-Nerve Fibres to the Elementary Muscular Fibres of the Mylo-hyoid Muscle of the little Green Tree Frog (Hyla Arborea). Drawn on the block by the Author, from a specimen magnified 1700 diameters (the first twenty-fifth made by Messrs. Powell & Lealand). The diameter of each muscular fibre corresponds to that of a human red blood-corpuscle.

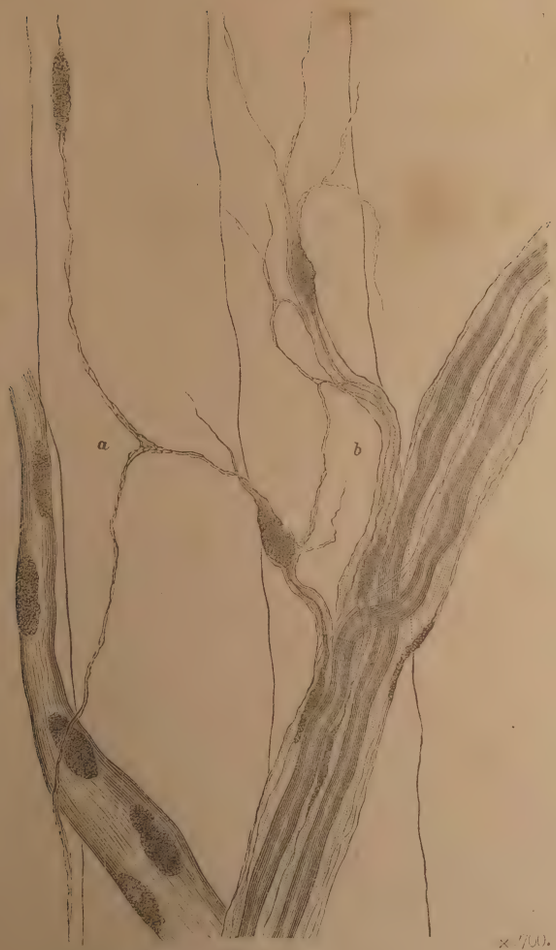
SCALE. $\frac{1}{10,000}$ of an English Inch $\frac{1}{25000}$ × 1700 diameters.

Fig. 1.



× 700.

Fig. 2.



× 700.

Fig. 3.

× 700.

Fig. 4.

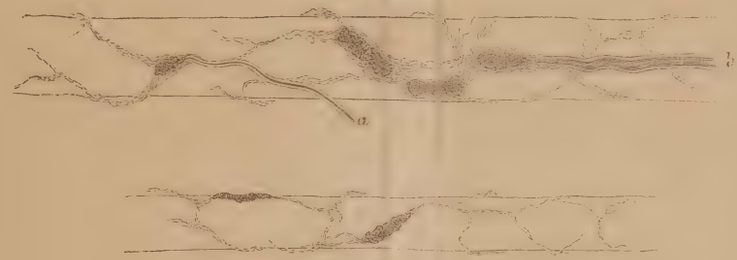
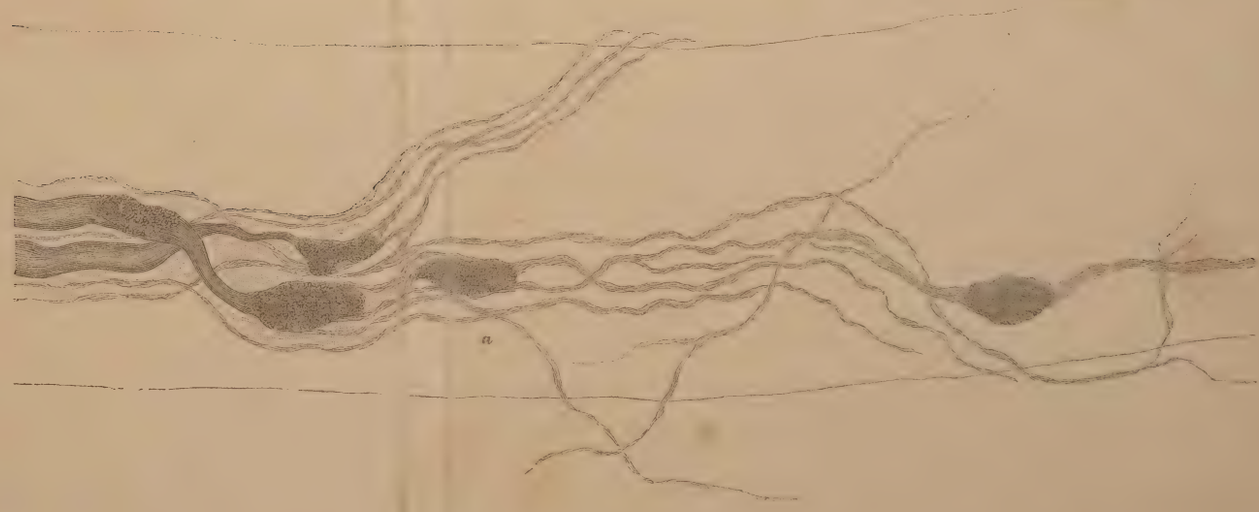


Fig. 5.

× 700.

Fig. 6.



×1800.

Fig. 7.

× 700.

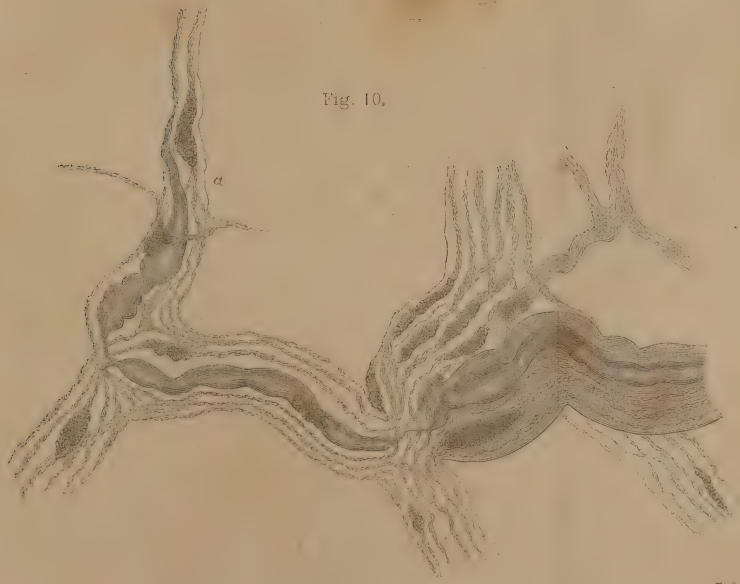
Fig. 8.

× 700.

Fig. 9.

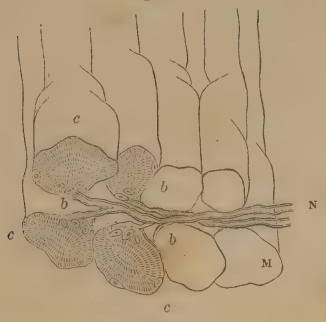
× 700.

Fig. 10.



× 700.

Fig. 1.



Kühne.

Fig. 2.

Kühne.

Fig. 3.

After Kühne.

Fig. 4.

× 215.

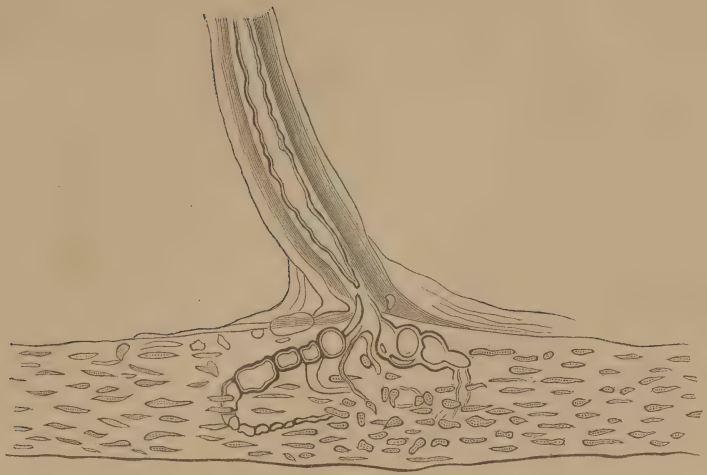
From the insect. L. S. B.

Fig. 5.

× 700.

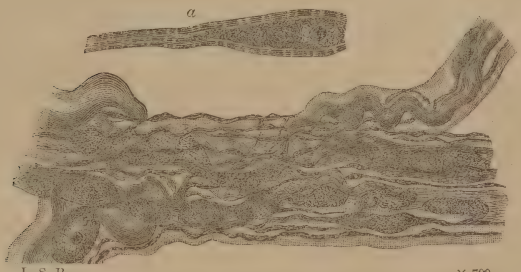
From the insect. L. S. B.

Fig. 1.



Kölliker.

Fig. 2.



L. S. B. × 700.

Fig. 3.

L. S. B. × 1000.

Fig. 1.

× 35.

Fig. 2.

× 420.

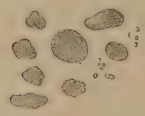
Fig. 3.

× 420.

Fig. 4.

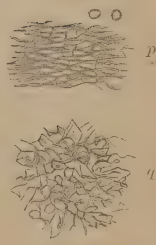
× 420.

Fig. 5.



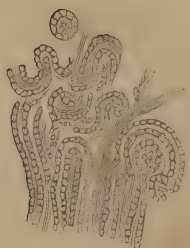
× 420.

Fig. 6.



× 670.

Fig. 7.

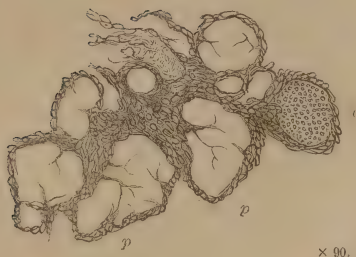


× 100.

Fig. 8.

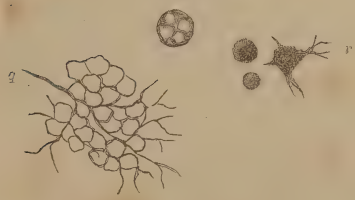
× 220.

Fig. 9.



× 90.

Fig. 10.



× 420.

Fig. 11.

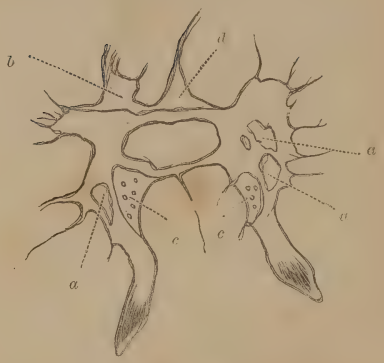


Fig. 12.

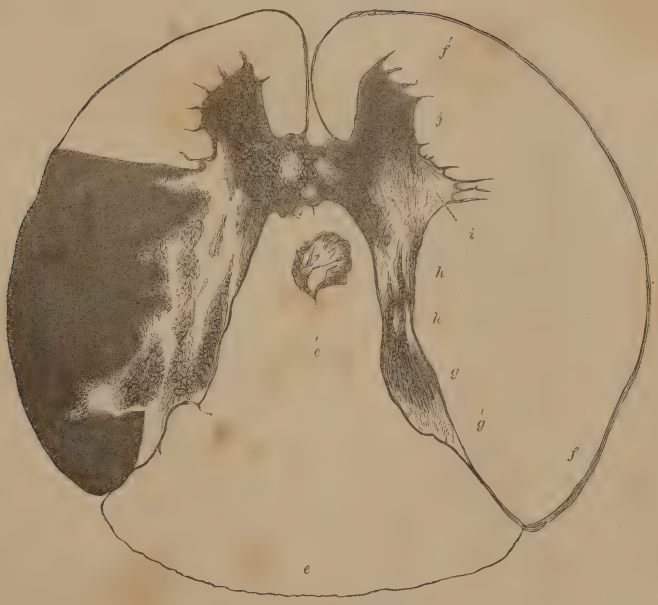
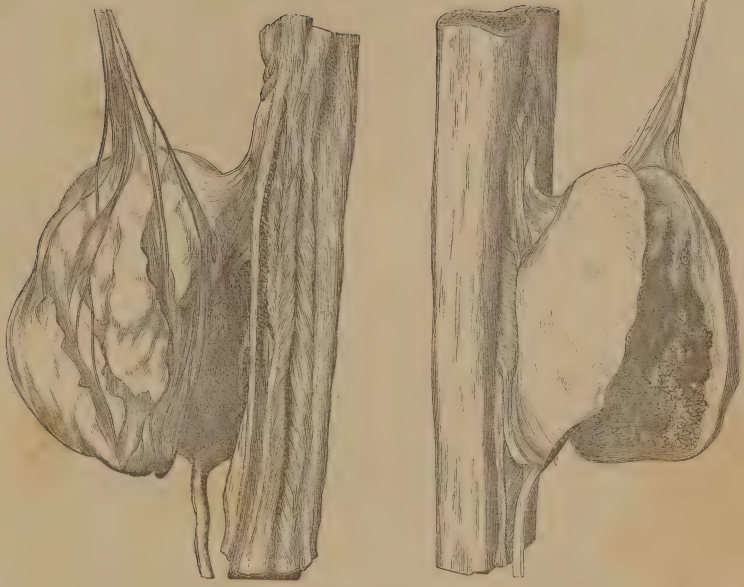


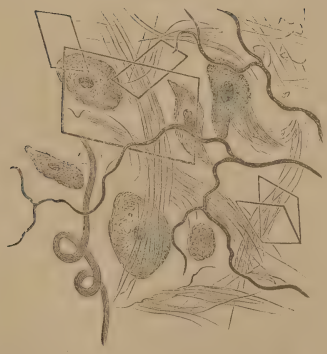
Fig. 1.

Fig. 2.



Neuromatous tumour. page 332.

Fig. 3.

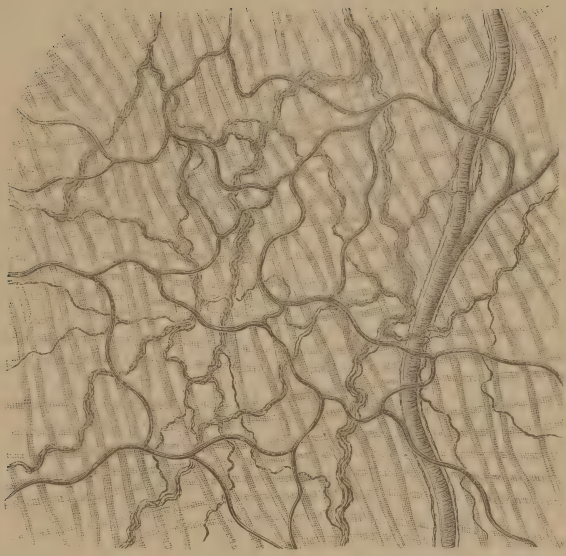


Structural elements of a small subcutaneous tumour. × 700. p. 334.

Fig. 4.

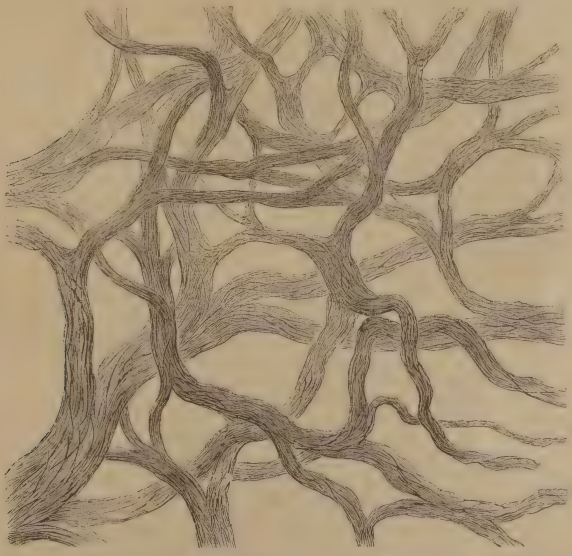
Fungi found in the tissue at the apex of the pyramids of the kidney *a* × 215; *b* × 700. p. 314.

Fig. 1.



Mylohyoid muscle of the Hyla, showing muscular fibres, capillary vessels, and net-works of dark-bordered nerve fibres × 130, and reduced to 65 diameters. page 315.

Fig. 2.



Networks, or plexuses of dark-bordered nerve-fibres, distributed near the free edge of the third eyelid of the common frog. × 220, and reduced to 110 diameters. p. 325.